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PARALYSIS OF THE VOCAL CORDS

A STUDY OF TWO HUNDRED AND SEVENTEEN MEDICAL CASES

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Paralysis of the vocal cords is a subject that has been studied widely, from both the clinical and the experimental standpoints. This may also be said of the limited amount of pathologic material available. Clinical observation of preoperative and postoperative cases of goiter has convinced us that the usually accepted textbook teaching, that is, the Semon law, does not seem to be substantiated. We have observed in such cases that a large percentage of the vocal cords following injury to the recurrent laryngeal nerve are in the median line position, and that a small percentage are in the cadaveric position. Follow-up data in later cases revealed that either the vocal cord returns to its normal function, or it pulls to the median line within a few months and remains there, as previously noted by one of us (Dr. New). A case has not been observed in which the vocal cord changed from the median line to the cadaveric position. A study has been made of 217 cases of paralysis of the cord examined in the Mayo Clinic. A review of these brings out certain facts.

HISTORICAL DATA

The Semon law, formulated fifty years ago, has not been entirely accepted, and objections to it have arisen from time to time.[1] Russell's work, which demonstrated that the abductor and adductor filaments existed in the recurrent laryngeal nerve in separate bundles of fibers, has not been repeated, although it has been attempted. It has further been shown experimentally that the fibers to the abductors lose their excitability more quickly than those to the adductors,[2] and various explanations of this have been offered. It was said that the nerve

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1. Critchley, MacDonald, and Kubik, C. S.: The Mechanisms of Speech and Deglutition in Progressive Bulbar Palsy, Brain **48**:492 (Sept.) 1925.

2. Donaldson, F., Jr.: The Function of the Recurrent Laryngeal Nerve, Am. J. M. Sc. **92**:93 (July) 1886. Semon, Felix, and Horsley, Victor: On an Apparently Peripheral and Differentiated Action of Ether upon the Laryngeal Muscles, Brit. M. J. **2**:405 (Aug. 28); 445 (Sept. 4) 1886.

fibers to the abductor muscles were similar to nerves to extensor muscles, following the general law that all extensor nerves are more vulnerable, also that the abductor muscles were not as old a group developmentally as the adductors. Grabower [3] stated that extension (abduction) is an elementary movement, whereas flexion (adduction) is a modified movement, a delicately graded action requiring more innervation. He found a much greater number of fibers in the nerves to the adductor muscles than in the nerve to the crico-arytenoid posticus muscle.

POSITION OF PARALYZED VOCAL CORDS

If the recurrent laryngeal nerve is injured, the vocal cords are ordinarily found in one of two positions, the median line or the cadaveric. Gerhardt [4] described both more than sixty years ago. Following injury to the nerve supply of the crico-arytenoid posticus muscle, abduction is lost, and the affected cord is found in the paramedian or median line. The tensor function persists and the cord is straight. During phonation, slight adduction may occur, presumably by the action of the arytenoid muscles, and a change in the voice may not be noticeable. The muscles receive a few fibers from the superior laryngeal nerve as well as their supply from the recurrent nerve.[5] The prospect of return of abductor function is said to be remote.[6] When bilateral abductor paralysis is present, laryngeal dyspnea may be marked and necessitate tracheotomy.

The term "cadaveric position," long considered inaccurate,[7] is generally understood to mean the position assumed following complete paralysis of the recurrent nerve, a position midway between the median line and the position of extreme abduction, or the expression of the complete relaxation of all the muscles. In the absence of any innervation from the recurrent nerve fibers, the cord is somewhat relaxed or bowed; it is, therefore, shortened, somewhat narrowed and depressed, lying at a lower level than normal. The arytenoid process lies farther forward than normally, and appears "fallen in." If, besides the paralysis of the recurrent nerve, there is loss of innervation from the superior laryngeal nerve, as may occur when the vagus is injured at the jugular

3. Grabower: Die diagnostische Bedeutung der Kehlkopflähmung, Berl. klin. Wchnschr. **1**:664 (April 7) 1911.

4. Gerhardt, C. G.: Studien und Beobachtungen über Stimmbandlähmung, Virchows Arch. f. path. Anat. **27**:68 and 296, 1863; quoted by Lermoyez and Ramadier, Presse méd. **1**:33 (Jan. 13) 1923.

5. Dilworth, T. F. M.: The Nerves of the Human Larynx, J. Anat. **56**:48 (Oct.) 1921.

6. Lermoyez, Marcel, and Ramadier, Jacques: La paralysie réspiratoire du larynx est un syndrome de syphilis, Presse méd. **1**:33 (Jan. 13) 1923.

7. Wright, Jonathan: History of Laryngology and Rhinology, ed. 2, Philadelphia, Lea & Febiger, 1914, p. 321.

foramen,[8] then the cord is in the true cadaveric position. If, however, only the recurrent laryngeal nerve is paralyzed, the vocal cord usually forms the median line position in about 90 per cent of the cases. In a small percentage of cases the vocal cord is in the cadaveric position. In the latter group, the affected cord that does not return to function pulls to the median line and is tensed by the action of two muscles, namely, the arytenoid and the crico-arytenoid muscles, which receive innervation from the superior laryngeal nerve. It may be possible that, even in the presence of complete paralysis of the larynx on one side, the cord may be pulled to the median line by the arytenoid muscle, which could act because of the innervation it receives from the opposite or sound side. It is well known that a vocal cord that no longer receives innervation through its recurrent laryngeal nerve may be found in the median or paramedian position, rather than in the cadaveric or intermediate position.[9] This fact was emphasized by one of us (Dr. New [9h]), who stated that following operations for goiter during which the recurrent laryngeal nerve was injured and the affected cord was in the cadaveric position, after several months it would swing to the median line with, incidentally, recovery from aphonia. The anatomic explanation of this is supplied by Dilworth.[5] It seems probable that because of the compensatory adduction that almost invariably follows, many cases of complete paralysis of the recurrent laryngeal nerve have been mistakenly diagnosed as of simple abductor paralysis. This may even have caused Semon to misinterpret the laryngoscopic picture in some of his cases, as it seems highly improbable that there should be incomplete recurrent paralysis in such a great number of cases as has been reported in previous years. Doubtless the fact that there are 4 separate nerves supplying the larynx accounts for the confusion concerning paralytic lesions here. Peripheral paralysis of any type is

8. New, G. B.: Laryngeal Paralysis Associated with the Jugular Foramen Syndrome and Other Syndromes, Am. J. M. Sc. **165**:727 (May) 1923.

9. (*a*) Grabower (footnote 3). (*b*) Ballance, Charles, and College, Lionel: Surgical Treatment of Paralysis of the Vocal Cord and Paralysis of the Diaphragm, Brit. M. J. **1**:553 (March 26); 609 (April 2) 1927. (*c*) Banks-Davis, H. T.: Paralysis of the Arytaenoideus in a Woman Aged 24, Proc. Roy. Soc. Med. **11**:163, 1918. (*d*) Berlin, D. D., and Lahey, F. H.: Dissections of the Recurrent and Superior Laryngeal Nerves, Surg., Gynec. & Obst. **49**:102 (July) 1929. (*e*) Johnson, George: Laryngeal Symptoms which Result from the Pressure of Aneurismal and Other Tumours upon the Vagus and Recurrent Laryngeal Nerves, Tr. Med.-Chir.Soc. London **58**:29, 1875. (*f*) Lemon, W. S.: Lesions Affecting the Vagus Nerve, M. Clin. North America **7**:293 (July) 1923. (*g*) Negus, V. E.: Some Disorders of the Larynx, Lancet **2**:581 (Sept. 19) 1925. (*h*) New, G. B.: The Larynx in Diseases of the Thyroid, Ann. Clin. Med. **1**: 262 (Jan.) 1923. (*i*) Taylor, E. W.: Pneumogastric Nerve, in Osler, William: Modern Medicine, Philadelphia, Lea & Febiger, 1910, vol. 7, p. 571. (*j*) Thomson, St. Clair: Classification of Neuroses of the Larynx, in Diseases of the Nose and Throat, New York, Cassell & Co., 1926, p. 580.

usually complete and involves all the muscles supplied by the affected nerve. This is especially well illustrated in the case of the facial nerve. The significant differentiating point between central and peripheral paralysis of the facial nerve is the amount of facial paralysis present. If paralysis is complete, the lesion is almost always peripheral, whereas only a lesion of the brain gives partial paralysis. The same probably holds true for the laryngeal nerves. Peripheral paralysis of a single nerve is probably always complete. The resultant laryngoscopic picture is easily explained by unopposed action of muscles supplied by the other laryngeal nerves. True peripheral abductor paralysis probably occurs only as a result of a lesion minute enough to involve only abductor fibers at their distribution in the larynx.

TABLE 1.—*Paralysis of the Vocal Cords*

Cause	Cases	Men	Women	Average Age, Years
Congenital lesions	3	1	2	17.0
Lesions of central origin				
Bulbar lesions	15	9	6	43.5
Tabes	9	8	1	44.1
Lesions of peripheral origin				
Toxic neuritis	6	5	1	42.5
Tumors of nasopharynx (jugular foramen syndrome)	13	10	3	49.0
Tumors of hypopharynx and esophagus	34	30	4	57.2
Syphilis of larynx	10	9	1	39.9
Tumors of neck, nongoitrous	4	1	3	40.7
Goiter, benign	32	10	22	42.8
Goiter, malignant	10	7	3	56.2
Trauma to neck	4	4	....	30.2
Mediastinal glands, 28 malignant; 1 inflammatory	29	19	10	42.5
Aneurysm, aortic	24	23	1	49.5
Aneurysm, innominate or subclavian	3	2	1	.....
Mitral stenosis and pericarditis	10	4	6	35.6
Tuberculosis	11	9	2	35.9
Total	217	151	66	.....

CAUSES OF PARALYSIS OF THE VOCAL CORDS

Congenital Origin.—Paralysis of the vocal cords of congenital origin is apparently rare, particularly among adults. We encountered 3 cases in which laryngeal symptoms had been present since birth. In all of them involvement of the vocal cords was bilateral (table 1).

The first patient was a girl, aged 10 months, who had been blue at birth and who had had dyspneic attacks with stridor about once a month since then. The vocal cords were flabby and fixed in the median line position. The aryepiglottic folds were not adjacent, as occurs in congenital laryngeal stridor. Spina bifida occulta was also demonstrated.

The second patient, a woman, aged 24, stated that her voice had been weak since childhood. Both vocal cords were completely fixed in the median line position. The left arytenoid cartilage was prolapsed, and the right cartilage overlapped it.

The third patient, a man, aged 26, had been hoarse all his life, but increasingly so for the last few months. His mother had also been hoarse all her life. Examination of the larynx revealed paralysis of the arytenoid muscles and of the cricothyroid muscles. The cords were irregular in outline and somewhat bowed and were incompletely adducted on phonation with a distinct gap in the interarytenoid region. Their free border presented irregular ellipses. This case suggested a bilateral cortical lesion.[10]

Lesions of Cortical Origin.—It is generally agreed that since the cortical center for the larynx is bilateral, a unilateral cortical lesion cannot produce paralysis of the larynx.[11] Semon and Watson-Williams expressed the belief that a bilateral cortical lesion might result in paralysis of the adductors, but Grabower concluded that even bilateral cortical lesions could not produce lasting paralysis, for other parts of the brain would help phonation vicariously.

Lesions of the bulb, it is said, are always responsible for laryngeal paralysis of organic or central origin.[11e] Such bulbar lesions may occur in progressive bulbar paralysis,[12] in syphilis,[13] in the apoplectic form of bulbar paralysis, in syringomyelia or in multiple sclerosis.[3] The laryngeal paralysis of progressive bulbar palsy rarely occurs until late and is usually bilateral.[14] Laryngoscopic findings apparently vary somewhat,

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10. Semon, Felix, and Watson-Williams, E.: Laryngeal Neurosis, in Allbutt, Clifford, and Rolleston, H. D.: System of Medicine, London, The Macmillan Company, 1909, pt. 2, vol. 4, p. 259.

11. (*a*) Lermoyez and Ramadier (footnote 6). (*b*) Taylor (footnote 9 i). (*c*) Hall, F. de H.; Ferrier, David, and Permewan, W.: Discussion of the Etiology of Unilateral Paralysis of the Recurrent Laryngeal Nerve, Proc. Roy. Soc. Med. (Sect. Laryng.) **6**:139, 1913. (*d*) Kelly, A. B.: Neurological and Mechanical Factors Underlying Immobility of the Vocal Cords: I. Neurological Aspect, Brit. M. J. **2**:678 (Oct. 15) 1927. (*e*) Thomson, St. Clair: Classification of Neuroses of the Larynx, in Diseases of the Nose and Throat, New York, Cassell & Co., 1926, p. 579. (*f*) Wright, Jonathan: History of Laryngology and Rhinology, ed. 2, Philadelphia, Lea & Febiger, 1914, p. 324.

12. (*a*) Semon and Watson-Williams (footnote 10). (*b*) Barwell, Harold: Laryngeal Paralyses and Their Diagnostic Value, Lancet **1**:1481 (June 3) 1905. (*c*) Collier, James: Upon the Relative Affection of Muscles in Progressive Bulbar Paralysis, Rev. Neurol. & Psychiat. **1**:525, 1903.

13. Lermoyez and Ramadier (footnote 6). Thomson (footnote 9 *j*).

14. (*a*) Barwell (footnote 12 *b*). (*b*) Semon, Felix: Clinical Remarks on the Proclivity of the Abductor Fibers of the Recurrent Laryngeal Nerve to Become Affected Sooner than the Abductor Fibers, or Even Exclusively, in Cases of Undoubted Central or Peripheral Injury or Disease of the Roots or Trunks of the Pneumogastric, Spinal Accessory, or Recurrent Nerves, Arch. Laryng. **2**:197, 1881.

for whereas certain observers[15] found bilateral abductor paralysis, others[3] found paralysis that was predominantly recurrent and partly adductor, and still others[16] thought that the adductors seemed especially liable. Critchley and Kubik pointed out that the Semon law has not been fully investigated for nuclear and supranuclear lesions of the laryngeal nerves.

In our series there were 24 cases of paralysis of the cord which could be attributed to intracranial lesions, of which 9 were syphilitic. The 15 nonsyphilitic cases included 4 cases of hematomyelia, 1 of thrombosis of the medulla, 4 of progressive bulbar paralysis, 1 of progressive muscular atrophy, 1 of arteriosclerosis of the central nervous system, 2 of inferior poliencephalitis, 1 of subacute combined sclerosis and 1 of pseudobulbar paralysis.

The paralysis of the vocal cord was bilateral in only 3 cases. The paralysis was of the abductor type in all. These 3 were cases of paralysis caused by progressive bulbar paralysis, pseudobulbar paralysis or subacute combined sclerosis.

In the 12 cases in which paralysis was unilateral, it was left-sided in 9 and right-sided in 3. In these 12 cases, the position of the affected cord was definitely noted 8 times. It was in the median line in 5 cases and in the cadaveric in 3; in these 3 cases there was hemorrhage or thrombosis of the medulla. Of the 5 cases in which the cord was in the median line position, the causes were hematomyelia in 1 case, inferior poliencephalitis in 1, progressive bulbar paralysis in 2 and progressive muscular atrophy in 1. An unusual type of incomplete paralysis was noted twice. In 1 case the lesion was probably arteriosclerosis of the central nervous system. The left cricothyroid muscle was affected, and the cord was soft, flabby, irregular and depressed, with a wavy outline and some loss of adduction. In the other case, probably due to poliencephalitis, the left cord seemed to move well at first, but apparently became fatigued and was then stationary in the median line.

Tabes.—The paralysis of the vocal cord in tabes is said to be usually of the bilateral complete abductor type,[17] but it may be unilateral,[11d] and there may be complete paralysis of one side with the crico-arytenoid posticus affected, or the internus of the opposite side.[3] Ivanow[18]

15. (*a*) Casselberry, W. E.: Recurrent and Abductor Paralysis of the Larynx: Diagnoses and Treatment, Ann. Otol., Rhin. & Laryng. **17**:607 (March) 1908. (*b*) Collier (footnote 12 *c*).

16. Critchley and Kubik (footnote 1). Hall, Ferrier and Permewan (footnote 11 *c*).

17. Grabower (footnote 3). Semon and Watson-Williams (footnote 10). Gleitsman, J. W.: Recurrent and Abductor Paralysis of the Larynx: Introductory Remarks on Anatomy and Physiology; Etiology of Paralyses of Central Origin, Ann. Otol., Rhin. & Laryng. **17**:587 (Sept.) 1908.

18. Ivanow, quoted by Hall, Ferrier and Permewan (footnote 11 *c*).

used this as a basis for an objection to the Semon law. In tabes a peculiar jerking abductor motion may occur.[19] The laryngeal crises of tabes may occur in a case in which the larynx is apparently normal.[20]

The condition in 9 of our cases could be ascribed to tabes. The paralysis was bilateral in 6 cases and unilateral in 3. In 5 cases the position of the cord was noted. It was in the median line in 4 cases and was cadaveric in 1 case. Tracheotomy was necessary in 1 case in which the cords were fixed in the median line. In 1 case of incomplete paralysis of both cords the peculiar jerky motion occurring in abduction, which was referred to before, was present. Three of the 9 patients had been having typical laryngeal crises.

Paralysis of Peripheral Origin.—Usually paralysis of the vocal cords is secondary to peripheral lesions, which may involve the vagus nerve at its exit from the jugular foramen, its superior laryngeal branch in the neck or its recurrent laryngeal branches in the neck or mediastinum. Lesions that may be responsible for paralysis are consequently numerous. It is said that aneurysm of the aorta is the most common of all causes.[11c]

Peripheral Toxic Neuritis.—Various toxic and infectious agents have been said to be responsible for paralysis of the recurrent nerve.[21] These included lead, arsenic, alcohol, atropine and the toxins of diphtheria, influenza or typhoid fever. It has been shown, however, that paralyses ascribed to such causes are usually of the unilateral abductor or recurrent type, which, according to Delavan [22] and Lermoyez and Ramadier,[6] would indicate that the hypothesis of peripheral neuritis is untenable. It was further suggested that many cases of fixed cords reported as laryngeal paralysis may be caused by ankylosis of the cricoarytenoid [23] joint; Tilley [24] reported 7 such cases, in all of which the fixed cord was on the left side. All the patients recovered. Other observers,[25] however, hold such causes to be rare. In 6 of our cases

19. (*a*) Bender, Lauretta: The Cerebellar Control of the Vocal Organs, Arch. Neurol. & Psychiat. **19**:796 (May) 1928. (*b*) Greene, D. C., Jr.: A Study of the Larynx in Tabes, Boston M. & S. J. **154**:97 (Jan. 25) 1906.

20. Greene (footnote 19 *b*). Fox, Noah: Laryngeal Crises of Tabes, Illinois M. J. **54**:66 (July) 1928.

21. Taylor (footnote 9 *i*). Thomson (footnote 9 *j*). Kelly (footnote 11 *d*). Barwell (footnote 12 *b*).

22. Delavan, D. B.: The Etiology of Paralysis of the Recurrent Laryngeal Nerves of Peripheral Origin, Ann. Otol., Rhin. & Laryng. **17**:631 (March) 1908.

23. Delavan (footnote 22). Wright, Jonathan: History of Laryngology and Rhinology, ed. 2, Philadelphia, Lea & Febiger, 1914, p. 325.

24. Tilley, Herbert: Mechanical Immobility of the Vocal Cords, Brit. M. J. **2**:681 (Oct.) 1927.

25. Thomson (footnote 9 *j*). Syme, quoted by Tilley (footnote 24).

it seemed probable that toxic neuritis might have been responsible for paralysis of the cord. The laryngoscopic findings were variable. The following are abstracts of these 6 cases:

CASE 1.—A man, aged 50, had used alcohol excessively for years; he had had increasing dyspnea for five months. Two years before he had had a paralytic stroke, and many years before he had had a chancre. He stated that even before the stroke occurred he had been told that his larynx was paralyzed. Examination revealed only total paralysis of the left cord and partial paralysis of the right.

CASE 2.—A man, aged 23, fourteen years before had bathed in snowy water, and shortly afterward pain in the side and hoarseness appeared, which had persisted. The left vocal cord was completely paralyzed.

CASE 3.—A man, aged 48, had had influenza a year before. For the last three months he had been hoarse, with occasional attacks of inspiratory dyspnea. Bilateral, incomplete (50 per cent) abductor paralysis was present.

CASE 4.—A man, aged 21, following an attack of measles eight years before, had been hoarse continuously. Five years later an attack of pneumonia had caused such severe dyspnea that tracheotomy was done; the tube had to be worn for the ensuing two years. Both vocal cords were fixed in the median line.

CASE 5.—A woman, aged 57, following scarlet fever forty-one years before, had been hoarse continuously. The left vocal cord was fixed in the cadaveric position.

CASE 6.—A man, aged 56, following diphtheria thirty-nine years before, had been hoarse continuously. The hoarseness was aggravated by infection of the upper part of the respiratory tract. Bilateral paralysis of the tensor muscles of both cords was present.

Syndrome of the Jugular Foramen.—A tumor situated in the nasopharynx often involves several cranial nerves at the exit from the jugular foramen and produces various combinations of paralysis of the cranial nerves.[26] The vagus nerve is often paralyzed, and consequently complete paralysis of the vocal cord is the rule. The affected cord is usually found to be in the cadaveric position.[27] In 13 cases a syndrome of the jugular foramen was caused by a malignant tumor and paralysis of the vocal cord. The paralysis was always unilateral, and was left-sided in 3 cases and right-sided in 10. The position of the affected cord was noted in 9 cases; in 5 it was cadaveric and in 4, median line. In the remaining 4 cases, the paralysis was apparently complete, since the cord was described as being completely fixed.

Hypopharynx or Esophagus.—Paralysis of the vocal cord, occurring as a result of carcinoma of the esophagus, supplied the data on which both Rosenbach and Semon based their law regarding paralysis of the cord fifty years ago.[28] Recurrent paralysis from carcinoma of the esoph-

26. Dilworth (footnote 5). New (footnote 8). Thomson (footnote 9 *j*).

27. New (footnote 8). Lemon (footnote 9 *f*)

28. Grabower (footnote 3). Semon (footnote 14 *b*).

agus occurs mainly among men with carcinoma of the upper third of the esophagus.[29] The involvement, usually left-sided,[6] may be bilateral.[30] The paralysis is said to be of the abductor type. In cases of bilateral paralysis, the lesion usually differs on the two sides; on one side it is recurrent; on the other, at first, posticus paralysis.[3] We observed 34 cases of proved carcinoma of the hypopharynx or esophagus which caused fixation of the vocal cord. In 3 cases both cords were paralyzed, and in the remaining 31 cases paralysis was unilateral. The right side was affected in 13 cases, and the left in 18. The position of the affected cord, noted in 9 cases, was median line in 7 cases and cadaveric in 2. In only 3 cases was there paralysis of the cord; in the remainder it was listed as fixed.

Syphilis of the Larynx.—In 10 cases paralysis of one or both cords was the result of tertiary syphilis of the larynx. Both cords were involved in 6 cases, and only one cord was affected in 4 cases. All the unilateral lesions were left-sided. The paralysis was usually of the complete abductor type, and tracheotomy was necessary in 2 cases. Not infrequently partial return of function followed subsidence of the acute process or treatment.

LESIONS OF THE NECK

Nongoitrous.—Four cases of paralysis of the cord were due to cervical tumors. These were a neurofibroma of the left carotid body, an osteochondroma arising from the first and second ribs, an adenocarcinoma, graded 3, and an indeterminate type of malignant growth that subsided under irradiation. As would be expected, the paralysis was unilateral in each case. The left side was affected in 3 cases, and the right in 1 case. The position of the affected cord was cadaveric in 3 cases, and median line in 1 case.

Goiter.—It is well known that enlargement of the thyroid gland, whether benign or malignant, may cause paralysis of the vocal cord.[31] An intrathoracic goiter is more likely to cause injury than one limited to the neck. Five of the 32 benign goiters in our cases were situated substernally. A malignant goiter is more likely to cause paralysis of the cord than benign enlargement of the thyroid gland. The left cord is more often affected. Bilateral paralysis before operation is rare.[9h] In 32 cases there was paralysis of the vocal cords as a result of benign goiter and in 10 cases as the result of malignant goiter. The principal differences between the two groups were the more advanced age of the patients with malignant disease, a shorter duration of the laryngeal

29. Sargnon, A.: Paralysies récurrentielles dans le cancer de l'oesophage, Arch. internat. de laryng. **7**:1044 (Nov.) 1928.

30 (*a*) Tilley (footnote 24). (*b*) Casselberry (footnote 15 *a*). (*c*) Guisez, J.: Malignant Disease of the Oesophagus, Brit. M. J. **2**:903 (Nov. 5) 1924.

31. Lemon (footnote 9 *f*). Kelly (footnote 11 *d*). Greene (footnote 19 *b*).

symptoms and a higher percentage of occurrence of completely paralyzed or cadaveric cords. The paralysis was unilateral in 40 cases and bilateral in 2. In the unilateral group, the left cord was affected in 24 cases and the right cord in 16. The positions of the affected cords in 21 cases were about equally cadaveric and median line. The position of the cord was median line in 8 cases and cadaveric in 6. In the malignant group the position was median line in 2 cases and cadaveric in 5. In 5 cases the paralysis from benign goiter was only partial. In 2 cases both cords were affected. A nonmalignant goiter caused bilateral abductor paralysis, and a malignant goiter caused fixation of the left cord and paresis of the right.

TRAUMA

Delavan [32] reported 2 cases of recurrent paralysis secondary to accidental trauma to the neck, the result of falls. The affected cords were shorter than normal and fixed in the paramedian line. In our series 4 similar cases were observed. In 1 case, a woman, aged 29, had been hoarse four months following an automobile accident in which she sustained a deep laceration of the neck. There was resultant paralysis of the left side of the tongue, and the left cord was fixed in the median line. Three other cases were more like those of Delavan. The patients were men. Hoarseness or aphonia had been present only a few weeks in each case following recent accidents. The first man had an automobile accident while driving with a pipe in his mouth. The right cord was fixed in the median line. The second man, while running, struck his throat against a boy's head. Total paralysis of the left cord resulted. The third man struck his neck against a wire while running. Both cords were injured. The right cord was completely paralyzed, and there was 50 per cent abductor paralysis of the left.

DISEASE OF THE MEDIASTINUM

Paralysis of the recurrent nerve may occur as a result of various pathologic intrathoracic conditions, among which may be mentioned malignant disease, either primary in the lungs or bronchi or metastatic from other regions of the body, Hodgkin's disease, inflammatory enlargement of the mediastinal nodes, aneurysm of the aorta or subclavian artery, cardiac lesions such as mitral stenosis with and without pericarditis and tuberculosis.

Twenty-nine cases of paralysis of the larynx were attributable to mediastinal disease, 28 of which included primary malignancy of the lung or bronchus, metastasis from carcinoma of the breast, pelvis or

32. Delavan, D. B.: Paralysis of the Right Recurrent Laryngeal Nerve from Accidental Trauma: Two Cases, Tr. Am. Laryng. A. **34**:114, 1912.

rectum and sarcoma (Hodgkin's disease). The paralysis of the cord was unilateral in each case. The left cord was more often affected, there being 22 left-sided and 6 right-sided lesions. In 20 cases the position of the cord was stated; 11 were cadaveric, and 9 were in the median line. The duration of laryngeal symptoms in the former group was six and four-tenths months. If the cords were in the median line, symptoms had been present only one and four-tenths months. Reexaminations were not possible.

One patient was seen with the left cord fixed following pneumonia. Clinical and roentgenographic examinations disclosed an apparently inflammatory node of the mediastinum and the results of an old lesion in the left side of the thorax from pneumonia.

Aneurysm of the Aorta.—This condition generally is considered the most common cause of paralysis of the left recurrent nerve. The paralysis is usually complete.[31] The right recurrent laryngeal nerve may be paralyzed by an aneurysm of the innominate [9f] or subclavian [3] artery. It is said that a very large aortic aneurysm may cause right-sided or bilateral recurrent paralysis.[33] Lermoyez and Ramadier stated the belief that usually paralysis of the vocal cord, more particularly the bilateral type, which in reality is caused by syphilitic lesions of the nucleus ambiguus in the bulb, is erroneously ascribed to aneurysm. In our experience, bilateral involvement of the vocal cords is not rare as a sequel to aortic aneurysm.

Of 24 cases of aortic aneurysm, the left cord was paralyzed in 19 and both cords in 4. In 1 case the right cord was paralyzed separately. Of the 20 unilateral cases, the position of the cord was noted in 11; in 7 cases it was cadaveric, and in 4 median line. Of the 4 cases of bilateral involvement of the cords, there were in each of 2 cases complete paralysis of the left cord and from 50 to 75 per cent abductor paralysis of the right cord; in a third case paralysis of both cords was equally complete, and in a fourth, the left cord was cadaveric, and the right was fixed in the median line. In the latter case an aneurysm involved both the aortic arch and the innominate artery. Tracheotomy was necessary in 1 case. In 2 cases there was an aneurysm of the innominate artery with resultant fixation of the right cord. In a third case an aneurysm of the subclavian artery resulted similarly.

Cardiac Lesions.—The explanation of the mechanism of the paralysis of the recurrent laryngeal nerve found associated with mitral stenosis has not been definitely agreed on. It was said by some that the nerve became compressed between the dilated left auricle and the arch of the aorta [34] or between the left pulmonary artery and the aorta or aortic

33. Johnson (footnote 9*e*). Hall, Ferrier and Permewan (footnote 11*c*). Barwell (footnote 12*b*).

34. (*a*) Grabower (footnote 3). (*b*) Ortner, quoted by Langeron, L.: Bruxelles-méd. **9**:163 (Dec.) 1928.

ligament;[35] others stated that the nerve became paralyzed as a result of traction on it by the aortic arch, which was dragged on by the enlarged heart,[11c] or from traction by the lung, which had been displaced by the dilated right ventricle.[36] Still others believed that inflammatory conditions in the pericardium or mediastinum are necessary.[37] The paralysis is usually on the left side,[11d] but may be bilateral.[34b] Recovery may occur when the circulatory condition improves [38] or if the paralysis is due to pericarditis or mediastinitis,[37a] when the inflammatory process subsides.

Ten cases of paralysis of the vocal cord could be ascribed to cardiac lesions. In 3 of these the diagnosis was adhesive pleuropericarditis, in 1 case associated with emphysema and in another with dilatation of the aorta and innominate artery. In the 7 remaining cases a diagnosis was made of chronic mitral endocarditis with stenosis, with a dilated or a dilated and hypertrophied left auricle. In 1 of the 7 cases there was concomitant aortitis. The left cord was always the one paralyzed, and this paralysis was of the complete recurrent type in 6 cases, as manifested by cadaveric cords. In 2 cases the affected cord was in the median line.

Tuberculosis.—Either recurrent nerve may become paralyzed as a result of tuberculosis, but the right nerve, because of its proximity to the apex of the right lung, is said to be particularly liable.[39] The nerves may also become involved by tracheobronchial lymph nodes near the hilum of the lung. The right nerve, being nearer the nodes, is also more liable to involvement than is the left nerve.[3] Recovery from paralysis of tuberculous disease [30b] is apparently the rule.

Tuberculous conditions of the thorax were the responsible factors in 11 cases of paralysis of the cord. In all cases the paralysis was unilateral; in 8 it was on the left side and in 3 on the right side, which is a contradiction of the belief of several observers, who found the right nerve more often paralyzed. The position of the cord, stated in 5 cases, was cadaveric in 3 and median line in 2. The 3 cadaveric cords were all left-sided, while 1 cord in the median line was on the left.

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35. Fetterolf, George, and Norris, G. W.: The Anatomical Explanation of the Paralysis of the Left Recurrent Laryngeal Nerve Found in Certain Cases of Mitral Stenosis, Am. J. M. Sc. **141**:625 (May) 1911.

36. Kraus, quoted by Langeron, L.: Bruxelles-méd. **9**:163 (Dec.) 1928.

37. (*a*) Adam, Alfred: Nervus recurrens Lähmung bei Mediastinitis, Arch. f. Laryng. u. Rhin. **27**:430, 1913. (*b*) Langeron, L.: Rétrécissement mitral; paralysie récurrentielle; syndrome de Claude Bernard Horner, Bruxelles-méd. **9**:163 (Dec.) 1928.

38. Brown, G. E., and Hempstead, B. E.: Paralysis of Recurrent Laryngeal Nerve Associated with Mitral Stenosis, J. A. M. A. **70**:4 (Jan. 5) 1918.

39. Grabower (footnote 3). Lemon (footnote 9*f*). Kelly (footnote 11*d*. Barwell (footnote 12*b*).

REEXAMINATION OF PARALYZED VOCAL CORDS

In 26 cases patients were reexamined after intervals varying from a few weeks to a number of years. The paralysis in these cases was due to various causes, and the duration and extent of paralysis also varied. It seems logical, however, to conclude from a study of these 26 cases that, in general: (1) a partially paralyzed vocal cord (so-called abductor type) may completely recover function; (2) a vocal cord fixed in the median line may completely recover, but usually remains in this position, and (3) a vocal cord fixed in the cadaveric position may completely recover, but usually swings to the median line within a few months and remains so.

Partial or Complete Recovery.—There were 6 such cases in our series. In 2 cases incomplete abductor paralysis (about 75 per cent) of the left vocal cord was caused by goiter. Thyroidectomy was performed, and when the larynx was examined two years later in 1 case and six years later in the other, the cords moved normally. In 3 cases paralysis was caused by syphilis of the larynx. The paralysis was bilateral in all cases; in 2 the cords were in the median line. In 1 of these cases tracheotomy was necessary. Examination of the larynx one year later in 1 case and two years later in the other revealed about 50 per cent of residual bilateral abductor paralysis. In the third case of this series the right cord had recovered; the left was unchanged. The sixth case was one in which the right vocal cord had become fixed in the median line because of a malignant cervical tumor. This was irradiated, and four months later the cord moved normally. The duration of the paralysis in these 6 cases, as determined from the history, varied from two to eight months, but in 1 case it may have been present as long as ten years.

Median Line Position.—In 12 cases the affected cord was in the median line and remained so. The cause of paralysis in these cases was benign goiter in 6, malignant tumor of the thyroid gland in 1, syphilis of the larynx in 1, a syndrome of the jugular foramen in 1, tabes in 2 and chronic pleuropericarditis in 1. The probable duration of the paralysis in these cases as determined from the history varied from ten days in the case caused by syphilis of the larynx to from two to five years in those cases caused by goiter, a syndrome of the jugular foramen and tabes. In 2 cases of goiter (one of these malignant) the paralysis had been present about eight months. The time interval between examinations varied from a few months to from one to fifteen years in cases of goiter, two months and one year in cases of tabes, one year in the case of a syndrome of the jugular foramen, four years in the case of syphilis of the larynx and two years in the case of chronic pleuropericarditis.

Change from Cadaveric to Median Line.—There were 6 such cases studied, and in 5 the lesion was unilateral and on the left side. In the sixth case, both cords were affected. The causes of paralysis were tuberculosis, 1 case; pseudobulbar paralysis, 1 case; cervical tumor, 2 cases, and goiter, 2 cases. The duration of paralysis as determined in 4 cases in which a history was given varied from a few months to a year, but in 1 of these it had probably been present for four years. The interval between examinations varied from nine months in 2 cases to one year, one and one-half years, seven years and thirteen years, respectively, in 4 cases.

No Change in Cadaveric Position.—There were only 2 such cases, in 1 of which the right cord was paralyzed as a part of the syndrome of the jugular foramen and which remained in the cadaveric position for eight years. One of us (Dr. New) has found that in his series of cases presenting the syndrome of the jugular foramen the vocal cord was always in the cadaveric position, thereby substantiating this finding. The other case was caused by goiter, and a change in the position of the right vocal cord had not occurred after two months, but will probably ultimately change to the median line as in similar cases.

It seems significant that the ultimate position of the cords in 18 of the 26 cases was in the median line. The cords were originally either in the median line or in the cadaveric position. None of those in the median line was later found in the cadaveric position; in some of these cases, however, the patients recovered. These were undoubtedly cases of incomplete paralysis of short duration.

In reference to the foregoing, another group of cases may be mentioned, namely, 39 in which paralysis of the vocal cord followed operation for goiter. About half of these patients had been operated on elsewhere; consequently, in these the laryngoscopic examination was usually made several months or years after operation. In the other cases, laryngoscopic examination was made soon after the operation. The affected cord at the first examination was usually in the median line, but in 12 cases it was cadaveric. Examinations were made at varying intervals following the first examination, from one month to nine years later except in 4 cases in which examination was made within three weeks after the first examination. Ultimately the cords were in the median line in 27 cases. Only 2 of the cords originally in the cadaveric position did not change to the median line. Improvement or recovery occurred in 10 cases.

Relation of Position of Cord to Duration of Paralysis.—In further support of the hypothesis that almost all vocal cords paralyzed as a result of recurrent paralysis will ultimately be in the median line, it was found that the duration of laryngeal symptoms was usually much greater in cases in which the vocal cord was fixed in the median line than it was in cases in which the cord was in the cadaveric position.

The duration of laryngeal symptoms was not obtainable in all cases, but in 43 in which the cord was in the median line the average duration of symptoms was seventeen months, and in 49 cases in which the cord was in the cadaveric position the average duration of symptoms was only ten and a half months. This would indicate that position in the median line usually accompanies paralysis of longer duration. Similar findings were noted in another group of 105 cases in which the cause of paralysis was indeterminate. In 24 cases the cords were in the median line and in 22 in the cadaveric position. Patients whose cords were in the median line had had laryngeal symptoms for an average of twenty and seven-tenths months, whereas those with cadaveric cords had had symptoms for an average of only four and a half months. As New and Lemon pointed out, cases of paralysis of the vocal cord from goiter are most satisfactorily studied, as far as paralysis of the recurrent laryngeal nerve is concerned. In spite of the fact that the recurrent laryngeal nerve is often severely injured in these cases, the paralyzed cord is predominantly in the median line. It may be cadaveric at first, but later is almost always in the median line. In a group of 123 cases of paralysis of the vocal cord secondary to operation for goiter, either here (34 cases) or elsewhere (89 cases), the position of the affected cord was noted in 97 cases, and in these it was median line in 88 and cadaveric in only 9. The average duration of associated symptoms with the cords in the median line was thirty-nine and a half months. In cadaveric cases symptoms had been present for an average of only five and seven-tenths months. This is further evidence that complete recurrent paralysis results in a median line position of the affected vocal cord.

Frequency of Paralysis of the Right and Left Cord.—It is generally agreed that the left cord is paralyzed about twice as often as the right.[40] This is apparently due to the more frequent involvement of the left cord by mediastinal lesions. The latter cause a large percentage of the cases of paralysis of the cord. Lemon stated that in the mediastinum the left recurrent laryngeal nerve is more often affected, as it is subject to pressure because of its central position and because of the anatomic design of the mediastinum and root of the neck. The right nerve is not so centrally situated, but is liable to tuberculous disease owing to its proximity to the pleura. Hall stated his belief that both sides are affected equally when paralysis is due to central causes or toxins or to malignant disease of the esophagus. He found the latter to be the most common cause of paralysis of the right cord.

In our series of 217 cases of paralysis of known etiology, both cords were affected in 32 cases and 1 cord was affected in 185. The left cord

40. Hall, Ferrier and Permewan (footnote 11 *c*). Barwell* (footnote 12 *b*).

was paralyzed in 127 cases, or more than twice as often as the right, which was paralyzed in 58 cases. This preponderance of left-sided paralysis was due largely to conditions in the thorax, namely, malig-

TABLE 2.—*Percentage of Causes of Laryngeal Paralysis (217 Cases) and Percentage of Left, Right and Bilateral Paralysis of the Cord from the Various Causes*

Cause	Cases				Percentage Total Paralysis in Each Disease Group		
	Left	Right	Bilateral	Total	Left	Right	Bilateral
Congenital lesions	....	....	3	3	.....	.....	100.00
Bulbar lesions	9	3	3	15	60.00	20.00	20.00
Tabes	2	1	6	9	22.22	11.11	66.67
Toxic neuritis	2	....	4	6	33.33	.....	66.67
Tumor, nasopharynx (jugular foramen syndrome)	3	10	....	13	23.08	76.92	.....
Tumor, hypopharynx or esophagus	18	13	3	34	52.94	38.23	8.83
Syphilis of larynx	4	....	6	10	40.00	.....	60.00
Tumor of neck (not goiter)	3	1	....	4	75.00	25.00	.....
Goiter, not malignant	17	14	1	32	53.12	43.75	3.13
Goiter, malignant	7	2	1	10	70.00	20.00	10.00
Injury to neck	2	1	1	4	50.00	25.00	25.00
Mediastinal lymph nodes	23	6	....	29	79.31	20.69	.....
Aneurysm (aortic)	19	1	4	24	79.16	7.05	13.79
Aneurysm (innominate or subclavian)	....	3	....	3	.....	100.00	.....
Mitral stenosis and pericarditis	10	....	....	10	100.00	.....	.....
Tuberculosis	8	3	....	11	72.72	27.28	.....
Total	127	58	32	217	58.52	26.72	14.76

TABLE 3.—*Percentage of Median Line and Cadaveric Positions in 132 Cases of Paralysis of the Cord from Various Causes*

Cause	Cases			Percentage Total Paralysis in Each Disease Group	
	Median Line	Cadaveric	Total	Median Line	Cadaveric
Congenital lesions	2	....	2	100.00	.....
Bulbar lesions	8	3	11	72.72	27.28
Tabes	4	1	5	80.00	20.00
Toxic neuritis	2	1	3	66.67	33.33
Tumor, nasopharynx (jugular foramen syndrome)	4	5	9	44.44	55.55
Tumor, hypopharynx or esophagus	7	2	9	77.78	22.22
Syphilis of larynx	6	2	8	75.00	25.00
Tumor of neck (not goiter)	1	3	4	25.00	75.00
Goiter, not malignant	17	9	26	65.38	34.62
Goiter, malignant	2	5	7	28.57	71.43
Injury to neck	2	....	2	100.00	.....
Mediastinal lymph nodes	9	11	20	45.00	55.00
Aneurysm (aortic)	5	8	13	38.46	61.54
Mitral stenosis and pericarditis	2	6	8	25.00	75.00
Tuberculosis	2	3	5	40.00	60.00
Total	73	59	132	55.30	44.70

nancy of the mediastinum, aortic aneurysm and cardiac lesions. The left cord, however, seemed even more vulnerable in some other conditions. The left cord was more often affected in cases of certain bulbar lesions, of tumors of the hypopharynx or esophagus and of goiter and even in cases caused by tuberculosis (tables 2 and 3).

This observation regarding the preponderance of paralysis of the left cord was further demonstrated in another group of cases. In reviewing our series of 217 cases of known cause, a fairly large group was encountered in which it was impossible to demonstrate any cause. There were 105 such cases that had been carefully studied for evidence of syphilis, toxic neuritis, arthritic lesions or lesions in the pharynx, larynx, neck, thorax or cranium. This difficulty in demonstrating the cause of paralysis of the vocal cord had been pointed out in other series of cases [11e] as varying from a third to a half of the cases.

In our group of 105, the average age of the patients was 43 1/10 years. Seventy-six patients were men. The average duration of laryngeal symptoms was thirty-two and eight-tenths months. Paralysis was bilateral in 8 cases and unilateral in 97. In unilateral paralysis, the left cord was affected in 67 cases and the right cord in 30. Again it may be noted that the left cord is affected more than twice as often as the right. The affected cords were in the median line or cadaveric position about an equal number of times. A similar preponderance of cases of paralysis of the left cord was noted in a group of cases in which paralysis followed thyroidectomy.

CONCLUSIONS

1. Usually paralyzed vocal cords in the cadaveric position change from this position to the median line within a few months, with return of the voice. If the condition is bilateral, dyspnea develops as the voice improves.

2. Vocal cords in the median line may return to complete function, but most of them remain in the median line.

3. None of the vocal cords observed in the median line change to the cadaveric position.

OSTEOMYELITIS OF THE SKULL ORIGINATING IN THE TEMPORAL BONE

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In this communication the cases of osteomyelitis of the skull which originate in any part of the temporal bone will be discussed. This group includes (1) cases that occur after, and as a result of, trauma; (2) hematogenous cases of osteomyelitis, and (3) extension cases which complicate acute and chronic inflammatory disease beginning in the middle ear or in other parts of the otologic apparatus.

Only those cases due to infection by the ordinary pyogenic bacteria (staphylococci, streptococci, pneumococci, etc.) are included, and no case of infection by the tubercle bacillus, by Spirochaeta pallida or by Actinomyces will be considered.

In discussing osteomyelitis of the temporal bone, no attempt will be made to include the subject of middle ear or mastoid disease as customarily understood among otologists and among internists except where elucidation of the subject and the text requires it. This phase of disease is beyond the subject of this communication.

The historical side of the subject of osteomyelitis of the skull in general has been described fully in another communication [1] and will not be repeated here. Suffice to say that the first mention of this subject was made by Pott [2] in 1768, and the first definite association of para-osseous cranial abscesses and disease of the underlying bone was made by Lannelongue [3] in 1879, although the relationship was suspected by Chassaignac [4] in 1859. The literature concerning osteomyelitis of the temporal bone is relatively small as compared with that concerning osteomyelitis of the skull in general and osteomyelitis of the skull complicating nasal accessory sinus disease in particular.

The records at Mount Sinai Hospital contain three proved instances of osteomyelitis of the temporal bone which fall into the group discussed in this paper; another very probable case has no postmortem

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From the Mount Sinai Hospital.

1. Wilensky, A. O.: Osteomyelitis of the Skull, Arch. Surg., in press.

2. Pott, Percival: Observations on the Nature and Consequences of Those Injuries to Which the Head is Liable from External Violence, London, L. Hawes, 1768.

3. Lannelongue, O. M.: De l'ostéomyélitis aiguë pendant la croissance, Paris, Asselin & Cie, 1879.

4. Chassaignac, quoted by Kallenbach: Beitr. z. klin. Chir. **128**:725, 1923.

proof and is not included in this communication. In addition, I have one case of osteomyelitis of the zygomatic process which I saw in consultation.

PATHOLOGY

The pathology of osteomyelitis of the skull in general has been extensively discussed on another occasion. Suffice to say here as follows:

Mild cases affecting the external table of the skull correspond in their pathology to osteomyelitis of the long bones limited to a relatively small area of the cortex. The circulation becomes shut off from a number of vessels in the external table and, depending on their number and on the extent of collateral circulation, a segment of bone is sequestrated and discarded. The sequestrated parts take the form of small flakes of bone, or thin segments of irregular shape. The suppuration which accompanies this gathers between the outer table and the pericranium and corresponds to a subperiosteal abscess of the long bones.

Mild cases affecting the internal table correspond in their particulars to those of the external table.

In more severe cases of osteomyelitis of the skull involving both the external and internal tables of the skull and in the diffuse and in the spreading varieties, the pathology is more extensive, and a dominant place is taken by the diploic network of veins. The pathologico-anatomic appearances of the lesions vary with the stage of the disease. In the early stages, the bone presents no abnormalities evident to the naked eye, although during life the diploe might perhaps show some congestion. In the next stage the diploe on section is seen to be markedly hyperemic and to be interspersed here and there with drops of pus. This is followed by the conversion of the medullary tissue of the diploe into granulation tissue bathed in pus, which, on section of the bone, exudes copiously from the cut surfaces. Thrombosed veins may be seen occupying the diploe. In the third stage the purulent secretion, finding its way to the outer and inner surfaces of the bony cranium by the vascular channels, or through minute openings formed by the resorption of bone tissue, collects to form abscesses between the bone and the pericranium on the one hand, and between the bone and the dura on the other—the para-osseous abscesses.

In the external variety of abscesses, the pericranium, raised off the surface by the accumulation, quickly undergoes dissolution, so that the collection of pus comes to lie in the subcutaneous tissues of the scalp. No delimiting abscess wall is formed, but the pericranial abscesses are, nevertheless, when they are multiple, quite discrete and separate from each other. These are the doughy or puffy swellings of the scalp, which form a striking clinical feature of the disease.

The dura shows little or no tendency to break down for considerable periods of time. Larger and smaller collections of pus gather in the extradural space which when sufficiently large may give rise to compression of the intracranial contents. The bone (external) wall of the abscess remains bare, but the dural (internal) surface becomes covered with shaggy granulations. Fine adhesions are usually demonstrable between the deep surface of the dura and the underlying arachnoid.

The fourth stage of the lesion is marked by more or less widespread destruction of the tables of the skull, the external usually giving way before the harder vitreous.

In favorable cases, in which healing and cicatrization become definitely established, however, the gaps made in the skull by the spontaneous, or operatively produced, loss of tissue are almost entirely filled up again by the formation of new bone.

Microscopically, early specimens show that the first changes consist in an invasion of the bone spaces by micro-organisms, with round cell infiltration of the medullary spaces, the walls of which, at this period, are still quite smooth, or are only just beginning to show some signs of lacunar absorption. The diploe is hyperemic and congested. Its consistency is decreased, and it contains small fungous growths and droplets of pus. It soon becomes grayish and contains sequestrums. They are generally numerous and small and may remain in the bone for a more or less long time, or may be eliminated. The alterations of the bone are especially seen on the external table.

The later changes, which may be regarded as secondary to the necrosis of the bone, consist in the replacement of the bone marrow by granulation tissue, with absorption, erosion and finally disappearance of the bony trabeculae of the diploe. The outer and inner tables withstand the eroding action till a still later period, so that extensive sheets of granulation tissue develop between the still intact, though lifeless, tables. The blood vessels contain thrombi more or less infiltrated with pus cells, and signs of perivascular inflammation accompany thrombosis when it progresses in advance of the general bone disease.

In cases in which general blood infection occurs, the viscera show lesions of a general infection (pyemia). The liver may be increased in size and may show amyloid degeneration. The spleen becomes hypertrophied. The lungs show nodules of hepatization and bronchopneumonia.

PATHOGENESIS

According to the pathogenesis involved, cases of osteomyelitis of the temporal bone can be classified as: (*a*) primary, (*b*) extension and (*c*) hematogenous.

Primary Cases.—This subject has been discussed on another occasion as regards osteomyelitis of the skull in general. Suffice to say here

that primary cases of osteomyelitis of the temporal bone can occur only as a result of an open wound in which the solution of continuity of tissue extends partially or completely through the thickness of the temporal bone. Under these conditions infection can be introduced directly into the temporal bone, and an osteomyelitis can be set up. Under modern conditions of therapy, this complication ought not to occur. While theoretically this form can occur, there is no record of such an occurrence at Mount Sinai Hospital, and I have seen no mention of such in the literature.

Extension Cases.—Extension forms of osteomyelitis of the temporal bone are all derived from foci of infection in the middle ear and mastoid cells.

Cranial osteomyelitis after middle ear and mastoid disease is encountered in the literature under two conditions: (1) as isolated and incomplete references to this association in the course of reports of cases of mastoiditis (Todd,[5] Harrison,[6] Pratt,[7] Druss and Friesner,[8] etc.), or (2) in larger communications (Eagleton[9] dealing (*a*) with the subject of nasal accessory sinus disease and its complications or (*b*) with the subject of cranial osteomyelitis. The sparsity of the literature is noteworthy because it calls attention to the actual small numbers of cases of cranial osteomyelitis that follow an otologic origin and to the relative abundance of this complication after nasal accessory sinus disease as compared with its occurrence after middle ear and mastoid disease. This is due both to (*a*) the essential anatomic conditions and to (*b*) the character of the manipulations during operation for mastoid disease.

(*a*) The squamous portion of the temporal bone, the petrous pyramid, the zygoma, the adjacent parietal bone and the occipital bone are the bony structures that are of interest. In looking at an articulated skull it is readily seen that the pneumatic portion of the temporal bone, which makes up the mastoid, is surrounded in the superior, anterior and posterior directions by a continuous area in which the diploe between the tables of the skull is at a minimum. In the flare of the squamous portion of the temporal bone the diploe is completely absent; some diploe is usually present in the root of the zygoma; anteriorly, where the bone articulates with the wings of the sphenoid, it increases somewhat in extent; toward the occipital bone, the cancellous structure begins to

5. Todd: J. Laryng., Rhin. & Otol. **24**:150, 1909.

6. Harrison: J. Laryng., Rhin. & Otol. **24**:628, 1909.

7. Pratt, E. L.: Laryngoscope **38**:409 (June) 1928.

8. Druss, J. G., and Friesner, I.: Pathways of Infection of Abscess of the Brain, Arch. Otolaryng. **13**:532 (April) 1931.

9. Eagleton, Wells P.: Cavernous Sinus Thrombosis, New York, The Macmillan Company, 1926.

become prominent. Here, however, it will be noted that the pneumatic structure of the mastoid is accomplished by plates of dense bone, and no true diploe exists. Posteriorly, also, where the temporal bone articulates with the occipital bone, the lateral sinus is lodged, and the diploe practically does not exist as its potential space is occupied by the venous sinus.

At the periphery of the temporal bone, where the bone thickens out to articulate with its neighbors, diploic structure begins to appear. In adults, as was pointed out previously, when the articulations as such disappear, the venous plexus contained in this diploe structure becomes continuous with that of its neighboring bones, the parietal, the occipital and the greater wing of the sphenoid.

The petrous portion of the temporal bone is composed of dense hard bone. It contains the auditory apparatus, the labyrinth and several canals for the transmission of nerves and vascular channels. In spite of its traditional hardness, a certain amount of cancellous (diploic) structure is found scattered in the very hard dense bone which makes up the bulk of the petrous pyramid and which makes up the various walls of the auditory apparatus, of the labyrinth and of various channels. Even though the middle ear and the mastoid cells are so closely associated with the pyramid, this dense bone seems to wall off the latter chambers very effectually from the little diploic structure contained within it.

Toward the base of the skull the apex of the pyramid lies in association with the basilar process of the occipital bone; the relationship resembles accurately that between the posterior part of the body of the sphenoid and the basiocciput.

(*b*) In operating for mastoid disease, the aim is to clear out all of the pneumatic portion. When the operation is efficiently done the entire cavity resulting from the operation is bounded by dense bone; the latter is a fairly efficient barrier to the transfer of any infection into the cranial diploe.

In association with otologic infection, the spread of the latter occurs in a number of directions. According to Neumann,[10] they are, in order of their frequency: (1) mastoid process; (2) fossa sigmoidea; (3) tegmen tympani et antri; (4) petrosa, including the labyrinth, and (5) posterior limb of the external semicircular canal and the carotid canal. According to Druss, from whom I am taking these facts and who gave me the experience of the otologic (Friesner) service at Mount Sinai Hospital, while the predilection for the cells of the petrous pyramid is fourth on the list, nevertheless, a petrositis is far more prevalent than

10. Neumann, H.: Die otitische Kleinhirn Abscess, Vienna, Franz Deuticke, 1907.

was previously believed. This has been demonstrated both clinically and histologically.

The foci of osteomyelitis, which are found to occur in the skull in association with otologic infection, roughly correspond to these various directions. The foci are as follows:

1. In the occipital bone. This is the commonest form: the infection spreads backward from the mastoid into the contiguous part of the occipital bone. This is usually interpreted as being an extraordinary extension of the mastoid cells, but in a certain number of cases, it is undoubted that there is a true extension into the diploe of the occipital bone. On the other hand, occipital involvement is extremely rare when a focus in the petrous pyramid spreads by contiguity to the basiocciput; this will be discussed subsequently in the section devoted to osteomyelitis in the pyramid.

2. In the zygoma. This is possibly next in frequency; it is usually found at the root and in the body of the zygoma, and is also sometimes interpreted as being an extraordinary development of the mastoid cellular structure. The lesion tends to localize itself in a relatively small area, and being very close to the floor of the middle fossa, it is one of the varieties in which perforation is found to occur into the interior of the cranium and to give rise to extradural abscess, meningitis, etc. Such a case was reported by Druss [11] from Friesner's service at Mount Sinai Hospital:

A boy, 6 years old, had headache and pain in the left ear, with spontaneous rupture of the drum. The symptoms continued. There were physical signs of meningitis. The spinal fluid contained meningococcus, and the blood culture was sterile. A roentgenogram of the mastoid area was negative. The boy was discharged with a diagnosis of meningococcic meningitis.

He was readmitted five months later with a seventeen day history of discharge from the ear for which myringotomy was done. Twelve hours before admission, frontal headache and vomiting developed. The spinal fluid showed gram-positive cocci, which did not grow on culture. The symptoms increased. Focal neurologic symptoms led to a diagnosis of temporosphenoidal abscess. The patient died.

Postmortem examination showed osteomyelitis of the inner aspect of the zygoma in the temporal bone and perforation of the bone through the adherent meninges. The remainder of the temporal bone was normal. There was a temporal lobe abscess.

3. In the squama. The thinness of bone in this locality is proverbial, and various forms of subdural abscess and meningitis are described. An illustrative case was reported by Eagleton.

The patient suffered from a deficiency in hearing. Sore throat developed, followed by increased diminution of hearing. Paracentesis was performed for

11. Druss, J. G.: Laryngoscope **41**:394 (June) 1931.

relief from pain. The maxillary antrum was drained. Right mastoidectomy was performed, and the patient did well thereafter.

"Cold" then developed followed by pain on the top of the head. The mastoid wound suppurated. There were no meningeal symptoms; fluid obtained at the lumbar puncture was sterile. Operation revealed extensive superficial necrosis of the squama just above the external auditory canal and zygoma, caries in the solid angle and serous meningitis. The sinus was free from infection.

Sepsis continued. A blood culture showed long chain streptococcus, the focus being in the shoulder. Death occurred.

Postmortem examination showed thrombophlebitis of the right jugular vein and bulb, a carious condition of the base of the skull over the sphenoid extending into the basilar process of the occipital bone, serous meningitis with an area of exudation, pneumonia and pericarditis.

4. In the petrous pyramid. Druss has studied these cases in Mount Sinai Hospital. Various paths have been described by Perkins,[12] Sears,[13] Chamberlin,[14] Maybaum [15] and others for the routes of extension of the infection from the middle ear and antrum to the pyramid. In the main, they are along the paralabyrinthine cells (above, below and on the side of the labyrinth), along the cells accompanying the eustachian tube, through the carotid canal, through the subarcuate fossa, along the superior and inferior petrosal veins, from an extension of an extradural abscess or from a combination of these. The infection as it spreads through the petrous pyramid may involve or avoid the labyrinth in its course. Other factors being equal, the more cellular the petrous pyramid the more ideal is the situation for the spread of infection.

According to Druss, the mastoid process, the petrous pyramid and the apex differ in their anatomic structure and in their pathologic picture. Usually the degree of infection is greater in the mastoid cells than in the paralabyrinthine cells, but the contrary may be true; there may be little or no infection in the mastoid cells, and the paralabyrinthine cells or the cells at the apex may be the seat of a localized collection of pus. Such cases have been reported by Mayer,[16] Karlefore,[17] Ulrich [18] and others. Druss demonstrated histologically that infection may subside in the middle ear and mastoid cavity but will increase in severity as it advances through the petrous pyramid. This bears out clinically the not too frequent occurrence of a dry resolving middle ear associated with an intracranial complication.

These anatomic considerations of osteomyelitis of the skull give the physical basis for: (1) its occurrence as a complication of middle ear

12. Perkins: Ann. Otol. **19**:692, 1910.
13. Sears: Pennsylvania M. J. **3**:844, 1910.
14. Chamberlin, W. B.: Atlantic M. J. **27**:566, 1924.
15. Maybaum, J. L.: Laryngoscope **30**:138, 1920.
16. Mayer, Otto: Monatschr. f. Ohrenh. **63**:930, 1929.
17. Karlefore, quoted by Druss (footnote 11).
18. Ulrich, quoted by Belinoff and Balan: Monatschr. f. Ohren. **64**:1185, 1920.

and mastoid disease; (2) its relative sparsity as compared with similar complications after nasal accessory sinus disease; (3) the essential similarity of the clinical symptom complex and course, and (4) similar associations with similar groups of intracranial complications.

The anatomic considerations outlined here give the physical basis for the very much lessened likelihood which exists in otologic conditions necessitating mastoidectomy of direct infection of the cranial diploe during the manipulations and steps of the operation. Unless the diploe is unusually well developed and extends well into and toward the field of a mastoid operation, it is difficult to understand how direct infection could mechanically be implanted into the diploe.

The following types of lesions are found:

1. Perforations. These are unusual. The cases at Mount Sinai Hospital included only one in which the perforation occurred in the neighborhood of the root of the zygoma. Theoretically, perforations ought to be found with disease in the squama.

2. Localized lesions. These form the bulk of the lesions found. Their localizations and physical appearance have been sufficiently indicated in the previous paragraphs. Practically all the remarks made here concern the localized type of disease.

3. Diffuse lesions. There are no cases of this kind in the records at Mount Sinai Hospital. When I speak to otologists about this type of lesion, every one is of the belief that they are very rare; and no one, when actually pressed for an example, can give me a specific instance of this kind. I have a slight recollection of having seen mention of such an experience in the literature, but I am unable to remember where and under what condition. I get the distinct impression that diffuse cases of osteomyelitis of the skull are extremely rare after otologic and mastoid disease.

The sequence of events in osteomyelitis of the skull of otologic origin is, for practical purposes, exactly like that in osteomyelitis of the skull complicating nasal accessory sinus disease. Both of these sets of organs are hollow chambers, either of a simple or of a complicated outline, lined by mucous membrane and subject to the same type of infection produced in similar ways; indeed, in many cases there is a "pansinusitis," including in this term an infection of all of the accessory structures which communicate with the nasopharynx. The bacteriology is similar; the essential pathology, the mechanism of production and the progression of the the lesion are similar.

The relative sparsity of cases of cranial osteomyelitis of an otologic origin is explained by the relatively efficient way in which the area of primary infection—middle ear and mastoid—is walled off from connection with the general cranial diploe and by the relative

sparsity of diploe in that part of the skull immediately adjacent to the primary area. Probably in those cases in which the osteomyelitis of the skull develops, the diploe is extraordinarily well developed and reaches downward toward the area of primary infection to a greater extent than is usual. Given a similar origin, with similar organisms, and similar pathogenesis and pathologic-anatomic lesions, it is to be expected that the clinical course will be similar in both types of cases—in those originating in the nasal accessory sinuses and in those originating in the otologic area.

The same considerations apply with equal force to the various intracranial complications that occur—to their variety, to their pathogenesis, to their clinical course, to their treatment and to the final outcome.

The following additional cases of osteomyelitis of the skull after otologic origin culled from the literature belong in this group:

HUNTER TODD.[5]—A 53 year old woman was seen with middle ear and mastoid disease; during mastoidectomy, an area of osteomyelitis as large as the palm of one's hand and extending into the occipital bone was exposed behind the mastoid. Secondary spontaneous sequestration of necrotic bone from the occipital area occurred. Later there was recrudescence of symptoms over the parietal bone; therefore operative revision of the wounds and of new areas was performed. A period of partial remission followed with later recrudescence of symptoms. The patient became gradually worse until death occurred. Postmortem examination showed typical wormeaten appearance of the cranial bones with areas where bone was absent (sequestrated areas).

HARRISON.[6]—A 25 year old man was seen with middle ear and mastoid disease; radical mastoidectomy was done, followed by a septic type of temperature. Revision of the wound showed thrombophlebitis of the lateral sinus. There was a continuation of symptoms. Subperiosteal abscess developed over the squamous portion of the temporal bone; also spontaneous sequestration and numerous abscesses under the scalp. The patient was operated on twenty times with final recovery.

GRUNWALD.[19]—A 63 year old man was seen with middle ear and mastoid disease; at mastoidectomy, no pus was found, but the cells contained granulations. Later an abscess developed in the posterior triangle of the neck; this was incised, and drainage was instituted. There was retention of pus, and on revision of the wound, bare bone was felt in the occipital region. On secondary revision, the occipital bone was removed as far as the inferior nuchal line—the seat of a necrotizing osteomyelitis.

PRATT.[7]—A boy of 5 years was seen with purulent middle ear disease; exploration of the mastoid was done because of stiffness of the neck and a turbid spinal fluid containing hemolytic streptococci. Revision was done because the condition did not improve; exploration of the intracranial contents at this time gave negative results. Death occurred. Postmortem examination showed necrosis of the anterosuperior part of the petrous pyramid and meningitis.

EAGLETON.[9]—A patient was seen who suffered from alcoholic periodic insanity. There was infection of the upper respiratory tract with severe pain in the head;

19. Grunwald, L.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **2**:139, 1922.

on the following day stiff neck and pain in both ears developed. There was bogginess over the mastoids. The patient had a high temperature and a cough, and was delirious and irrational. Two blood cultures were negative. Culture from the ear revealed diplococci. The right mastoid was explored, and an abscess was found in the suboccipital region due to suppurative osteomyelitis of the occipital bone; there was pus in the right mastoid. Antemortem blood culture (four hours) showed streptococcus. Death occurred.

Postmortem examination showed sphenoid sinus suppuration; occipital edema; superficial abscess below the mastoid; deep abscess in relation to the upper four cervical vertebrae; purulent thrombophlebitis of the right lateral sinus extending to the torcular and into the jugular vein, involving the right superior and inferior petrosals, cavernous and anterior and posterior circular veins, and also the left cavernous and superior longitudinal sinuses. Other changes were incidental to the general infection in the viscera.

EAGLETON.[9]—The patient suffered from left chronic otitis media with acute exacerbation, accompanied by chills and high temperature (107 F.). Mastoidectomy revealed sclerosed bone and thrombophlebitis of the lateral sinus. The blood culture was negative. The septic type of temperature continued; operation was performed on the jugular bulb (Grunert's operation); there was no bleeding from the inferior petrosal sinus. A blood transfusion was done. A second blood culture was negative. Lumbar puncture revealed the fluid under slight pressure; the cell count was 28, the globulin was slightly increased, Fehling's solution was reduced and there were no organisms. The symptoms continued until death.

Postmortem examination showed marked jaundice; a small extradural abscess of the posterior surface of the left petrous bone, with a local area of necrosis near the posterior semicircular canal, the dura being separated from the bone; a suppurating jugular bulb and pus in the left lateral sinus extending to the torcular vein; thrombosis of the left superior and inferior petrosal veins; thrombosis of the right cavernous sinus and of the basilar plexus and anterior spinal veins passing through the foramen magnum.

KALLENBACH.[20]—A man of 33 years, four years before his present illness suffered a tangential gunshot injury of the right frontal protuberance with a defect in the skull. Eight days before admission he became ill with a mild grip. Since then he had had a boring pain in the right temple. After about six days, the swelling and pain increased. He was in a good condition of nutrition. The skin was slightly icteric, the tongue coated and dry, the sensorium slightly impaired. He would not raise his head; even the slightest movement caused intense pain. The temperature was 38.2 C. (100.7 F.), and the pulse rate 98. On the right frontal protuberance there was a small defect in the bone through which pulsation could be seen. No pain was felt on pressure over the frontal bone. There were doughy swelling and redness of the soft parts over almost the whole right temporal bone; the swelling was very sensitive to pressure. The ear and the mastoid process were normal. As his condition grew worse, operation was performed on the third day. The right temporal bone was exposed through a horseshoe incision. One place about the middle of the temporal bone was denuded of periosteum. Pus came from the bone at this place in small drops. The bone was chiseled open over an area about the size of a five mark piece and found interspersed with small abscesses. The dura was intact. The soft parts were sutured and a tampon inserted. Four days after the operation the patient's

20. Kallenbach: Beitr. z. klin. Chir. **128**:725, 1923.

mind was clear, and the pain had decreased considerably. The wound looked well. The tampon was replaced by a drain. On the eighth day, the general condition was good, but there was slight pain. On the twelfth day, the condition was worse. At times the patient was in a slight stupor; there was increased redness at the lower angle of the wound. On the thirteenth day, there was intense pain in the region of the third to fifth lumbar vertebrae. There was slight swelling there, but no redness; the pain on pressure was intense. On the fourteenth day the pain in the spinal column was greater; the swelling and pain on pressure were more intense. The wound in the head was normal; the general condition became worse. On the sixteenth day an incision was made at the site of greatest sensitiveness on pressure over the spinous processes of the third to fifth lumbar vertebrae. On incision of the soft parts, thick pus was found and the spinous processes were rough. They were clipped off with forceps, and the wound tamponed. Bacteriologic examination, like that of the wound in the head, showed Staphylococcus pyogenes-aureus. The general condition improved immediately after the operation; the process in the spinal colum did not proceed any farther, and the wound in the head healed. On the thirty-fifth day, the patient was discharged with the wounds healed and no symptoms.

Hematogenous Cases.—Attempts have been made to isolate a group of cases of osteomyelitis of the temporal bone originating in the otologic apparatus and to distinguish them from the general run of cases of mastoiditis of otologic origin.

A report of 266 cases of streptococcic osteomyelitis of the temporal bone was made by Boyd-Snee [21] in 1922. A great many facts were given, out of which one can extricate the following: (1) The osteomyelitis is always associated with a middle ear infection or with an exacerbation of it; (2) invasion occurs by continuity of structure or by hematogenous paths; (3) the streptococcus is always present; (4) the term used is "uncomplicated" osteomyelitis of the temporal bone, and (5) the mastoid is apparently uninvolved; (6) the symptomatology is that of mastoiditis; (7) the usual intracranial complications are likely to occur—thrombophlebitis, meningitis, brain abscess, etc.

In the discussion of Boyd-Snee's paper (Breitenbach, Bulson [22]), there was not entire agreement with the conclusions of Boyd-Snee. There seems to be a strong possibility that many cases of mastoiditis that are unusually widespread have been included in the 266 cases reported by Boyd-Snee.

Frey attempted to differentiate cases of ordinary disease of the middle ear and of the mastoid from cases of osteomyelitis of the temporal bone unassociated with otologic infection. His differential diagnosis depends on the following criteria: (1) particularly rapid development of the manifestations; (2) rapid deterioration of the general condition from the beginning with high temperatures, prostration,

21. Boyd-Snee, H.: J. Indiana M. A. **15**:147, 1922.

22. Bulson, A. E.: Osteomyelitis of the Frontal Bone as a Complication of Frontal Sinusitis, J. A. M. A. **86**:246 (Jan. 23) 1926.

anorexia and restlessness; (3) the presence of a retro-auricular swelling with especial reference to its early appearance, size and tendency to extend rapidly beyond the mastoid limits; (4) the virulence of the infection; (5) frequency of involvement of the facial nerve and of disturbances of the vestibular apparatus; (6) the operative appearance—(*a*) the tendency to necrosis, (*b*) the presence of foci close by in the zygoma, squama or pyramid, or at a distance by way of the diploic veins; (7) rapid progress of the disease, and (8) involvement of other than the lateral sinus. Frey [23] cited the following case:

A boy, aged 4 years, presented a retro-auricular swelling behind and below the mastoid and extending to the occiput and into the muscles of the neck. At operation there was widespread necrosis, and the osteitis was found to extend into the spongy cancellous tissue of the zygoma and into the occipital bone. A thrombosis of the lateral sinus with a surrounding abscess was present also.

Four days after operation there were present chemosis of the upper eyelids and exophthalmos; the temperature was 105 F. The spinal fluid was negative, the blood culture was sterile, and the pus showed staphylococcus. Eight days later jaundice developed and death occurred.

I have no doubt that cases of osteomyelitis of the temporal bone of this type—and most probably they are of hematogenous origin—occur from time to time. The chances are good that these differentiating criteria which Frey proposes are sound, and they are confirmed by Wittmaack.[24] But it would take exceptionally sound and clever clinical sense to make the differentiation unless evidence of a hematogenous infection in general is undoubted and beyond question. One of the points that would carry weight with me would be the presence of Staphylococcus aureus as the provocative and etiologic organism for the infection.

A group of cases occur in which a focus develops in the petrous portion of the temporal bone and in which there is no evidence of preceding middle ear disease or of mastoid involvement, or in which the middle ear alone is involved; sometimes the middle ear manifestations appear some time after the evidences of pyramid disease. The discussion always centers in these cases around the question of hematogenous infection. My own impression is that many of these are of hematogenous origin. There have been several of such instances in the otologic service at the hospital but usually the available data are somewhat insufficient to enable one to express an incontrovertible opinion. A very pertinent case is the following:

A 62 year old colored man entered the hospital (service of Dr. Baehr and Dr. Freisner) with an unimportant history except for occasional gas pains during the past three or four years. His present illness began four weeks before

23. Frey, G.: J. Laryng. & Otol. **40**:308 (May) 1925.

24. Wittmaack, in Henke and Lubarsch: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12.

admission with an initial shaking chill followed by attacks of pain in the lower left side of the chest, not increased by respiration, and accompanied by continued high temperature and daily severe chills. Physical examination revealed an acutely ill man with some impaired mobility of the left leaf of the diaphragm, bilateral costovertebral tenderness, more marked on the right, and marked tenderness over the liver. The blood pressure was 130 systolic and 90 diastolic. The hemoglobin was 82 per cent; the white blood count, 22,000 with 86 per cent polymorphonuclears. The Pirquet test was positive.

This clinical course continued in the hospital, and a week after admission a complete exploratory laparotomy was done, but nothing abnormal was found. The wound healed by primary union.

The patient continued to run the same septic course as before the operation. The Wassermann test was negative. Blood cultures were persistently negative. Agglutinations for typhoid and melitensis also were negative. A search for malarial parasites revealed nothing. Prostatic smear revealed some pus cells but no organisms. A roentgenogram of the chest on two occasions was negative. Roentgen examination of the stomach and duodenum was negative. Normal urine persisted throughout the patient's stay in the hospital, and an intravenous pyelogram, taken to rule out some renal infection such as a carbuncle, also revealed nothing. A leukocytosis of from 20,000 to 30,000 white cells with a polynucleosis of 90 per cent persisted. The general condition of the patient gradually deteriorated, and the only physical sign obtainable after operation was persistent marked tenderness over the liver.

Three weeks after admission, aspiration of the liver was performed. No organisms were obtained, and culture was negative. On the same day, following the aspiration, the patient had a severe shaking chill and a rise in temperature to 104 F. Examination shortly thereafter revealed definite signs of meningeal irritation; a spinal tap was performed, cloudy fluid being obtained that contained 4,000 cells and 76 per cent polymorphonuclear cells; smear and culture of the latter were negative. Daily examination of both ears revealed no changes. The sepsis and the meningeal signs remained unchanged, and the spinal fluid gradually improved; about eight days later the latter was clear and contained only 12 cells. The next day a thick discharge developed in the right ear; the ear drum was flat anteriorly, and only a small amount of fairly thick discharge was present in the inferior sulcus. Smear and culture of this pus revealed a pneumococcus type III. A roentgenogram of the mastoids was negative.

Two days after the ear began to discharge, a spinal tap was again made and revealed a cloudy fluid with 2,500 cells, 78 per cent of which were polymorphonuclear cells. Bronchopneumonia preceded death by about forty-eight hours.

Postmortem examination revealed chronic purulent osteomyelitis of the petrous portion of the temporal bone (right) with bone abscess, acute purulent basilar meningitis, carbuncle of the left suprarenal with suppuration of the posterior wall of the lesser sac and phlebitis of the splenic vein, thrombophlebitis of the portal vein with phlegmonous suppuration of the liver, bronchopneumonia of both lower lobes of the lungs, acute purulent bronchitis, parenchymatous degeneration of the viscera, acute congestion of the lungs and the liver, atherosclerosis with calcification of the aorta, hypertrophy of the left ventricle of the heart and cysts of the kidneys.

The notes of the case are deficient in very valuable and pertinent bacteriologic data. Nevertheless, it seems evident that this entire clinical entity is a hematogenous general infection with undemonstrable bac-

teremia in which metastatic foci were present in the suprarenal gland (a carbuncle) and in the petrous portion of the temporal bone. This opinion is concurred in by the otologic department (Friesner) and by the medical department (Baehr) at Mount Sinai Hospital.

ASSOCIATION OF OSTEOMYELITIS OF THE PYRAMID AND OF THE SPHENOID

There is a marked clinical similarity between disease of the pyramid and of the sphenoid bone. Both of these lie in the same anatomic position with reference to the base of the skull and the adjoining intracranial structures. Perforations occur in the pyramid as well as in the sphenoid, and there is the same predilection for extradural abscess and meningitis; brain abscess also occurs. A characteristic symptom complex for pyramid disease is Gradenigo's syndrome—involvement of the gasserian ganglion and of the abducent nerve—but as the syndrome has been found to occur without bone change, the connection is not absolute.

The intimate association between the petrous pyramid and the sphenoid bone is well illustrated in the case reported by Gelanze:[25]

> In this patient there was a ten year history of a chronic otorrhea and chronic otitis media which followed influenza. The present recrudescence began two months before admission with pain, fever, increased discharge, considerable retro-auricular swelling and the physical signs of meningitis including turbid lumbar fluid. A mastoid operation was done, and following this the manifestations diminished.
>
> One week before death the symptoms reappeared with increased meningeal signs. The local mastoid wound and the middle ear were not in a satisfactory condition (fungous granulations, cholesteatomas, etc.), and there was necrotic and sequestrating bone. The symptoms increased and facial paralysis developed. Following a sudden aggravation of symptoms, death occurred.
>
> The postmortem anatomic diagnosis was seropurulent meningitis of the left temporal and cerebellar fossa, and necrosis of the petrous bone, the sphenoid bone and the sella turcica, with perihypophysitis.

On careful study of this case, I agree with Gelanze that the infectious process, produced by germs of considerable virulence with a marked necrotizing power, had in the first place attacked the meninges through the tegmen antri, giving rise to meningitis which was promptly circumscribed by the local reaction. In spite of the rather extensive mastoid evacuation at the first operation, the osteomyelitic process became promptly propagated to the perilabyrinthine cells and to the labyrinth itself, thus permitting the germs again to attack the cerebellar fossa by way of the saccus endolymphaticus and to give rise to a new meningitic process. At the same time, in spite of the second inter-

25. Gelanze, C.: Valsalva **6**:86, 1930.

vention, and the complete petrotympanic evacuation, the necrotizing osteomyelitic process rapidly advanced toward the petrous bone, and after only two days resulted in sequestration of the entire facial segment, followed by paralysis of the facial nerve; the entire acousticovestibular nerve bundles underwent necrosis. On reaching the extreme mesial limit of the petrous bone, the osteomyelitis, by way of the periosteal vessels and lymphatics, next attacked the body of the sphenoid bone and became diffused to the sella turica, giving rise by its vicinity to a perihypophysitis which remained without evident clinical symptoms. The extension of the necrotic process and the considerable absorption of the toxic substances produced in large amounts in the infectious focus were the principal causes of the patient's death.

ROENTGENOGRAPHIC EVIDENCE OF OSTEOMYELITIS OF THE SKULL

Roentgenographic evidence of osteomyelitis of the skull, when once the lesion is established, is generally similar for all classes and varieties of cases no matter what the etiology or pathogenesis is. This, as is generally true in all cases of osteomyelitis, is not available at the beginning of the development of the lesion but appears relatively late. The evidence is available under two conditions:

1. In localized forms of disease. This takes the form of well demarcated sequestrations.

2. In the diffuse variety. There is a characteristic motheaten appearance of the bones of the skull.

Roentgenographic evidence is especially demonstrable and available with lesions in the vault. Lesions at the base of the skull, no matter what their roentgenographic morphology may be, are not demonstrable at all or are demonstrable with extreme difficulty.

The roentgenographic evidence of mastoiditis as ordinarily seen is not included in this discussion.

THE OCCURRENCE OF GENERAL INFECTION IN OSTEOMYELITIS OF THE SKULL

Cases with the clinical signs of a general infection can be found in each of the pathologic and clinical groups outlined in this paper as follows: (*a*) as the original general infection to which the skull lesion is a metastatic lesion, (*b*) as a result of a primary infection of the bone tissue of the skull in civil or military injuries, (*c*) as a secondary phenomenon in extension cases of osteomyelitis of the skull and (*d*) accompanying intracranial complications, especially meningitis and sinus thrombosis.

COMPLICATIONS

In cases of general blood infection (sepsis, septicemia, etc.), metastatic lesions are found post mortem in the various organs of the body; they are especially common in the kidneys, the liver and the lungs. Metastatic lesions such as these (i. e., kidney, liver, lungs) give no dominating picture and are lost in the general symptomatology, both subjective and objective, of the clinical picture.

The development of the intracranial complications and the clinical course during their development have in most instances followed a fairly definite order. The first stage is comprised in the infection of the skull bones. The second stage is marked by the occurrence of an external pachymeningitis; an extradural abscess usually results.

The third stage is a progression of the second, and, according to most of the observers, is produced fairly rapidly. A subdural infection occurs, and arachnoiditis and meningitis result.

In the further spread of the infection beyond these limits, the process spreads to the intracranial venous sinuses and to the brain. Thrombosis of the large sinuses, especially the cavernous sinus and the superior longitudinal sinus, is commonly seen in the published case reports. Brain abscess may occur at any stage of the development or in the absence of many of the stages herein outlined.

The majority of the intracranial complications occur in accordance with the following mechanisms: (*a*) by contact because of contiguity of structure, (*b*) by vascular and lymphatic connections and (*c*) by contact with secondary complication, usually a discrete extradural abscess separated and at a distance from the region of the main focus of cranial osteomyelitis.

OTITIC MENINGITIS

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After the development of streptococcic meningitis one is usually powerless to stop or allay it. Kolmer, Eagleton, Kopetsky and others are advocating its anticipation with premeningitis therapy. The alert otologist should have a thorough knowledge of pathologic processes and a broad experience in otology to evaluate premeningitic signs and symptoms and to prevent the development of meningitis before or after mastoidectomy.

It is impossible without too lengthy discussion to review the anatomy and physiology of the meninges, but a few of the pathologic processes that excite meningitis must be mentioned in order to understand premeningitic symptoms. Otitic meningitis usually develops from an extradural or an intrameningeal abscess in the middle or posterior fossa, thrombotic infection, embolic infection, rupture of an abscess of the brain, fracture of the skull through the tegmen or mastoid process or purulent labyrinthitis.

It is well to bear in mind that the meninges, especially the dura, are more resistant to infection than the intact bone and that adhesions and localized collections of cerebrospinal fluid tend to localize the infection if it has invaded the meninges. The extension through the normal-appearing bone and dura is often by thrombophlebitis without macroscopic necrosis of the bone. An extradural or intrameningeal abscess in the tegmen area of the middle fossa is the most difficult extension to diagnose, because it gives pressure in a fairly silent area of the brain. Significant findings of lesions of the middle fossa are: segment-shaped contractions of the visual field, mild changes in the cerebrospinal fluid, a slightly elevated temperature and headache of such a character that it will be relieved when there is a profuse discharge from the ear.

A deep extradural or intrameningeal abscess of the middle fossa has a more definite symptomatology. Irritation of the gasserian ganglion gives pain in the face, behind the eye and often in the teeth; Gradenigo's sign is not often present, but is possible; there is a continued septic temperature (from 2 to 4 degrees); the recurring discharge from the middle ear after the mastoid has been thoroughly operated on, the discharge from the eustachian tube from rupture of the cells of the tip and some

mild changes in the spinal fluid point rather definitely to suppuration of the petrous portion of the temporal bone. If these symptoms, especially the orbital pain, fever and recurring discharge from the ear, finally disappear, one should not be misled, for meningitis may occur later.

Extradural or intrameningeal abscesses in the posterior fossa are most often from suppuration of the petrous tip, and in my cases the operative finding was an extensive pneumatization of the mastoid process. The symptoms of involvement of the posterior fossa are a recurring discharge from the middle ear after operation, with a clean mastoid wound, progressive deafness of one or both sides, a possible involvement of other cranial nerves, facial, auditory, glossopharyngeal or abducens, a rise in temperature of a few degrees, a slightly increased cell count and globulin, an increased spinal fluid pressure and possibly pressure symptoms of the gasserian ganglion.

Depending on the size of the extradural or intrameningeal abscess, certain pressure symptoms may or may not be produced, such as headache, insomnia, restlessness, clouded sensorium, rising blood pressure, vomiting, slow pulse, cervical rigidity, palsies of the cranial nerves, choked disks, positive Babinski, Brudzinski and Kernig signs and ankle clonus.

Direct extension from a lateral sinus thrombosis rarely occurs and, in addition to the signs of an extrameningeal or intrameningeal abscess of the posterior fossa, has the antecedent picture of sinus thrombosis.

Fracture of the skull through the tegmen, mastoid or petrous pyramid, through an infected ear or mastoid, is a potential forerunner of meningitis because of the infection of the blood clot that accompanies the fracture. If the fracture is through the canal of the ear, cleaning the canal or disinfectants in the canal may directly infect the clot in the meninges and set up meningitis.

Rupture of a cerebral or cerebellar abscess may produce meningitis. Abscesses of the temporal lobe often rupture into the ventricle; those of the cerebellar lobe, into the basal cistern. The signs and symptoms must be kept in mind, but will not be reviewed here.

Purulent labyrinthitis is a potential source of meningitis and causes nystagmus to the opposite side, decreased hearing, vertigo and vomiting, and the caloric test does not elicit a reaction. An operation must be performed on the labyrinth immediately if meningitis is to be avoided, especially if there are an elevation of temperature and evidence of sepsis.

REPORT OF CASES

CASE 1.—Claude Peters, aged 43, was admitted to Epworth Hospital on April 25, 1927, with the diagnosis of meningitis. Ten days previously he had sustained an injury to the head, but there was no fracture; he had been away from work for twenty-four hours. Vertigo, nausea, vomiting and hiccups developed one week later. Fever and delirium started shortly before he was taken to the hospital. There was a history of chronic otitis.

Examination revealed a pulsating, profuse, purulent discharge from a fairly large perforation of the posterior inferior quadrant of the right ear-drum. The temperature was 105 F.; the pulse rate, 102, and the respiratory rate, 30. There were rigidity of the neck, bilateral Kernig and Babinski signs, horizontal rotatory nystagmus to the right (same side), small pupils, normal eyegrounds, a negative caloric test and increased cerebrospinal fluid pressure. Spinal fluid findings were: 4+ globulin and 1,180 cells, with gram-positive diplococci.

Mastoidectomy was performed. The mastoid cells were necrotic and were filled with pus and granulations. There was no demonstrable labyrinthine fistula. A Neuman labyrinthine operation and exposure of the posterior fossa through the labyrinth were done. Turbid spinal fluid welled up into the wound. The dura of the middle fossa appeared normal; the sinus was exposed. Daily irrigations with saline solution and weak mercurochrome-220 soluble by spinal and cisternal puncture through the wound were used after operation. The patient died on the third postoperative day. The spinal fluid was free from organisms and contained very few cells on the day of death.

At autopsy, no increased tension of the dura was found, and the cerebral and cerebellar convolutions were free from exudate. The pia-arachnoid was moderately injected on both sides. No exudate was found at the base, but some increased redness was present over the surface. The pia over the cerebellum was slightly edematous. The dura covering the right temporal bone and middle fossa showed no gross evidence of inflammation or exudate.

Comment.—This was a case of chronic otitis with a history of injury and the development of labyrinthitis in which a remarkable resistance of the meninges to infection was noted at autopsy. The patient was not seen until the meningitis with organisms was present. Eagleton has since shown that the spinal fluid does not tolerate mercurochrome.

Case 2.—Richard Smith, aged 6 years, was admitted to Epworth Hospital on Nov. 14, 1929, with the complaint of otitis media, bilateral recurrent attacks, the last being of two weeks' duration, continued pain and fever; the white blood cells numbered 13,150; the hemoglobin was 80 per cent.

Simple, bilateral mastoidectomy showed an enormous pneumatic mastoid with granulations and free pus, of the coalescent type. The sinus and dura were not exposed. During the following three weeks, a continued rise in temperature and pain in the right side of the face and teeth finally resulted in a secondary bilateral operation. Although the dura and sinus were exposed, no evidence to account for the symptoms was found. The pain in the face continued, and the temperature ranged from 98 to 102 F. On December 9, there was a positive Kernig and Babinski sign on the left for a few hours. On December 10th, the spinal fluid showed a slightly increased pressure, with 20 cells (lymphocytes) and 2+ globulin. On December 12, rigidity of the neck, a temperature of 104 F. and a positive Kernig and Babinski sign were present; the cerebrospinal fluid contained 3,000 cells and streptococci. Before death on December 16, the right pupil was contracted, and the left dilated.

Autopsy showed a large amount of fibrinous exudate over the base, blocking the foramina Magendi and Luschka. The right ventricle was filled with thick purulent fluid. In reflecting the dura of the middle fossa over the tip of the petrous pyramid, an area of necrosis of the bone was found. On section, this area was found to be necrotic, with infiltration into the dura just opposite the gasserian ganglion.

Comment.—This was an extensive case of pneumatic, acute mastoiditis with continued pain and fever after operation, with pain in the face and teeth on the right side as the localizing sign. A few weeks after this boy died I heard Eagleton read his paper entitled "Unlocking the Petrous Pyramid" in Chicago.

CASE 3.—Helen Elliott, aged 9 years, was seen on Feb. 28, 1931. Two weeks after the onset of acute otitis, mastoiditis developed. The temperature was 101.6 F.; there were pain, tenderness, a profuse discharge and a sagging canal wall. Mastoidectomy revealed a large abscess of the pneumatic, coalescent type. The dura of the middle fossa was normal. A stormy postoperative course followed, with fever and recurring discharge from the middle ear, pain in the eye and face, a clean mastoid wound and an increase in nonfilament polymorphonuclear neutrophils. The temperature became normal, and the patient was symptom-free until April 23. On that day, meningitis developed, with 2,160 cells and streptococci in the fluid. The mastoid was then reopened; the dura of the middle fossa and the sinus appeared normal. The anterior surface of the petrous pyramid was exposed as far as the gasserian ganglion, but necrosis was not found. The patient died on May 7.

Comment.—In this case the continued fever, pain in the face, clean mastoid wound and recurring, pulsating discharge from the middle ear pointed to suppuration of the petrous tip as the focus of the meningitis.

CASE 4.—David Peterson, aged 9 years, was admitted to the hospital on June 11, 1929. He had had otitis media for ten weeks and postauricular swelling for three weeks. One week prior to admission, a normal and subnormal temperature, vomiting, drowsiness, vertigo, falling to the left, nystagmus to the left, contralateral deviation of the eyes, weakness of the muscles on the left, intention tremor, loss of coordination on the left, bilateral choked disks and a positive Babinski sign on the left was noted.

Mastoidectomy revealed a pneumatic, suppurative, coalescent mastoiditis; the sinus and dural plate were intact. A cerebellar exposure was made on the left, and 6 cc. of green pus was evacuated. The next day meningitis appeared and death occurred.

Autopsy revealed granulations over the petrous tip in the posterior fossa, 0.5 cm. external to the internal auditory meatus. The abscess cavity in the cerebellum was empty.

Comment.—This cerebellar abscess, with every classic sign, was easy to diagnose. Meningitis was precipitated by the operative intervention. The dural exposure should have been incised and packed before the abscess was evacuated.

CASE 5.—Edward Szuba, aged 12 years, white, was seen on March 29, 1929, with the complaints of nausea and vomiting. He had had chronic otitis for ten months. Four days before admission he had had severe pain, a profuse discharge from the right ear, nausea, vomiting, chills and fever.

Examination revealed a rational boy, with a rigid neck and positive Kernig and Babinski signs. No palsies of the cranial nerves were demonstrable. There was a profuse, purulent, pulsating discharge from a large perforation in the right ear-drum. There was no local or roentgen evidence of mastoiditis. The

spinal fluid contained 770 cells; it was turbid, but there were no organisms. During the next five days, the discharge from the ear was not so profuse and not pulsating. The Babinski and Kernig signs and the cervical rigidity disappeared. On April 2, the ear was dry, and the patient felt good and wanted to go home. There were no localizing signs, but the white blood cells numbered 19,300.

On April 3, the patient began to vomit again, and on April 4, symptoms of meningitis were marked, with cloudy fluid and streptococcus organisms in the spinal fluid. The Queckenstedt sign was positive for jugular block.

On April 6, mastoidectomy was performed. Pneumatic coalescent mastoiditis, a lateral soft and caseous sinus plate and thrombosis were found. The dura of the middle fossa was normal.

On April 7, the patient died.

Comment.—Subsidence of the purulent meningitis should not have influenced me to refrain from operative intervention, as it was known that it was an otitic meningitis that had been walled off. This type of case, without organisms, has a fairly good prognosis if the local focus can be drained. After the organisms were found in the fluid, the prognosis was much more grave. The extension was apparently through the sinus, and this should have been attacked surgically before the second exacerbation.

CASE 6.—Ida Williams was seen on Feb. 4, 1930, in the service of Dr. C. E. Savery. There was a history of acute otitis of ten days' duration. She had had pain in the ear, headache and vomiting for the past two days. The vomiting continued, and on February 6 she had convulsions.

Mastoidectomy was performed on February 7, and revealed a pneumatic, coalescent mastoid; the dura was not exposed. The spinal fluid showed 110 cells and short chain cocci. On February 8, there were continuous convulsions, with death.

Autopsy showed a red, injected dura over the tegmen, and although the osseous tegmen was intact, it was also red. There was an intrameningeal abscess; the pia was red; the convolutions here were flattened.

Comment.—The vomiting, pain in the ear, headache and stupor indicated meningeal irritation, and the dura should have been exposed immediately. As Alexander said, whenever there are headache and vomiting one should look beyond the mastoid, for mastoiditis will not cause these signs. If the middle fossa had been exposed at operation, the dural changes would have been seen.

CASE 7.—A boy, aged 5 years, was seen by Dr. Wygant in 1927. During the preceding ten months he had had chronic otitis, with the last exacerbation three months previously. A fracture of the skull five days prior to admission, through the petrous portion of the temporal bone, resulted in the drainage of a bloody spinal fluid from the canal of the ear. The wound was treated by a surgeon with mercurochrome. On the fifth day, rigidity of the neck, chills, fever and positive Kernig and Babinski signs ushered in meningitis; 1,300 cells in turbid fluid with streptococci were found on lumbar puncture.

Mastoidectomy showed a sclerotic type of mastoid, with granulations and free pus in the antrum, and also a fracture through the tegmen antri and a laceration of the canal of the ear. The patient died of meningitis.

Comment.—Compound fractures through unaffected ear canals should not be infected by irrigation or medication applied to the canal. Infected ears that are fractured should have operative drainage and dural exposure to prevent infection of a dural clot in a sealed space.

The seven cases reported showed otitic, premeningitic symptoms that were fairly accurate precursors of the meningitis that resulted. Because of the different methods of extension to the meninges, the symptoms varied and should probably be the subject of separate papers rather than presented with the entire symptomatology under one heading. However, because I wish to stress the importance of the meningitic prodromal symptoms to be looked for in every case of otitis, I am unable to divide the subject into separate entities, as all symptoms must be considered in an examination of the individual patient with otitis. Only one set of symptoms is usually present, depending on the avenue of approach from the ear, but all avenues with their accompanying symptom complex must be visualized if meningitis is to be prevented and if the incidence of this complication is to be lessened.

SUMMARY

Labyrinthitis, the syndrome of involvement of the petrous tip, increased symptoms of intracranial pressure and fractures through infected ears are precursors of meningitis. Intact bone is not as good a barrier to infection as the dura and meninges. The meninges do not tolerate mercurochrome. Pain in the face, eyes and teeth, together with sepsis and Gradenigo's syndrome, indicates suppuration of the petrous tip. At operations on abscesses of the brain, one should expose and open the dura without attacking the abscess until adhesions have walled off the exposed normal brain and meninges. A subsiding purulent, otitic meningitis without organisms should call for careful operative intervention, because the intracranial extension has localized itself and may easily get beyond control again. When there are intracranial symptoms at operation, it is more important to expose the dura than the sinus.

Compound fractures through infected middle ears and the mastoid should be considered potential meningitis. Treatment of the ear canals with mercurochrome or irrigations is contraindicated in compound fractures with lacerations of the canal.

CONCLUSIONS

The otologist should be alert to all premeningitic signs and symptoms, and should have a knowledge of the methods of intracranial extension from the middle ear and a wide clinical and autopsy experience to be able to prevent fulminating cerebrospinal otitic meningitis.

PRIMARY MALIGNANT TUMORS OF THE LOWER THIRD OF THE TRACHEA

REPORT OF A CASE WITH SUCCESSFUL TREATMENT BY ELECTROFULGURATION AND DEEP X-RAYS

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"The lower portion of the trachea is very rarely the seat of tumors," stated Ewing.[1] An examination of the literature reveals that there are on record but twenty-four cases of carcinoma of the lower third of the trachea. D'Aunoy and Zoeller [2] were able to collect only nineteen cases in a survey covering a period extending through 1929. Since that time there have been but five other cases reported. A perusal of the clinical records of the Johns Hopkins Hospital reveals the fact that the case that I shall present is the only one of primary carcinoma of the lower third of the trachea seen in that institution during its forty-two years of existence. Correctly enough, it could be argued that, while the previously mentioned figures would lead one to believe that tracheal tumors were rare, the actual state of affairs might be, as Chevalier Jackson declared when he saw his first case, "such are not uncommon but simply undiscovered." However, nothwithstanding the more widespread practice of endoscopy during the past few years, it cannot be denied that primary malignancy of the lower third of the trachea is certainly not frequently encountered. As to the sex and age incidence of carcinoma of the lower part of the trachea, Breslich [3] found that it is almost twice as frequent in men as it is in women, and that most of the tumors occur within the age limits of 30 and 70 years.

Concerning their etioliogy one can only theorize. Some maintain that they develop from cell rests, while others favor the theory of metaplasia. Simmel [4] believed that the bifurcation was the most frequent

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From the Department of Oto-Rhino-Laryngology of the Johns Hopkins Hospital.

1. Ewing, James: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, p. 853.

2. D'Aunoy, Rigney, and Zoeller, Adelaide: Primary Tumors of the Trachea, Arch. Path. **11**:589 (April) 1931.

3. Breslich, P. J.: Squamous Cell Carcinoma of the Trachea, J. Cancer Research **14**:144 (March) 1930.

4. Simmel, E.: Zur Kasuistik des primären Carcinoms der Trachea, Arch. f. Laryng. u. Rhin. **24**:449, 1911.

site of tracheal carcinoma and considered that at this point the mucous membrane was most exposed to trauma. D'Aunoy and Zoeller [2] have found that the middle third of the trachea, which is more immovable and more protected than either the lower or the upper thirds of the organ, is less frequently the site of malignant processes. They have found that the posterior wall, which is richer in glandular structures than either the anterior or the lateral wall, is most often attacked. In this connection the work of Strauss [5] on the lymphatics of the trachea is of interest. He, as reported by Broman,[6] has brought out the follow-

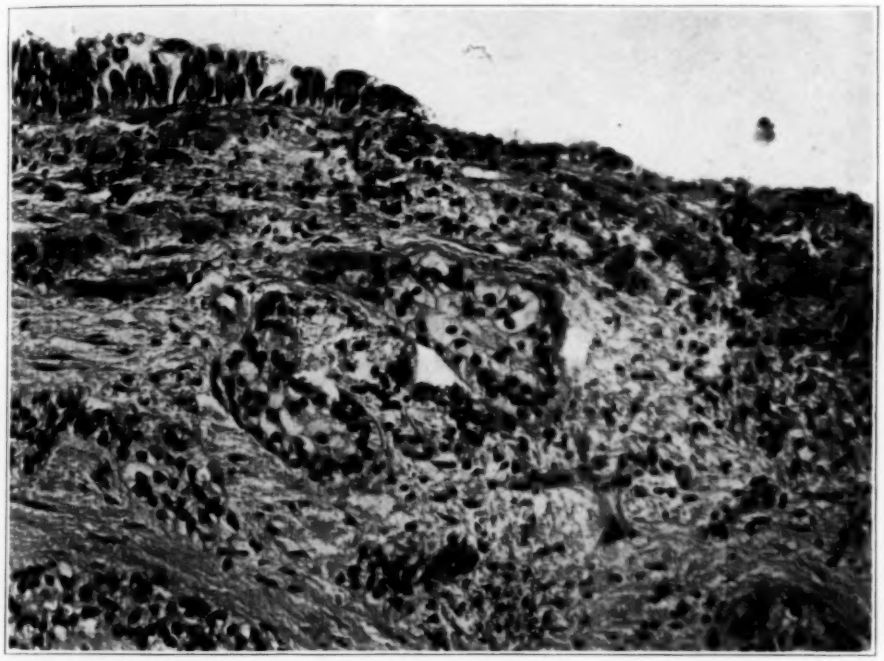

Fig. 1.—Low power view of tissue removed from a tracheal neoplasm.

ing points: The superficial network of fine lymph channels is divided into segments by the cartilaginous rings. Absorption from the mucous membranes of the trachea (when there is no inflammatory condition present) is most active in the upper and lower thirds, and least active in the middle. The tendency of the lymph drainage at any given point is toward the posterior wall and thence to the contiguous lymph nodes. There are, nevertheless, minor intersegmental capillaries and posterior

5. Strauss, J. F.: The Intimate Lymphatics of the Trachea, Ann. Otol., Rhin. & Laryng. **31**:715 (Sept.) 1922.

6. Broman, J. R.: Primary Carcinoma of the Trachea with Report of a Case and Review of the Literature, J. Cancer Research **8**:394, 1924.

plexuses by which there is fairly free anastomosis. The horizontal collecting vessels are more numerous and of a larger caliber in the region of the bifurcation.

REPORT OF A CASE

History.—A white man, aged 49, a carpenter, was seen in the department of oto-rhino-laryngology at the Johns Hopkins Hospital on Oct. 30, 1930. He complained of exertional dyspnea, which was progressively increasing. This condition had been present during the preceding fifteen months. He had never noted any edema or cyanosis. Occasionally there was "phlegm" in the throat which caused

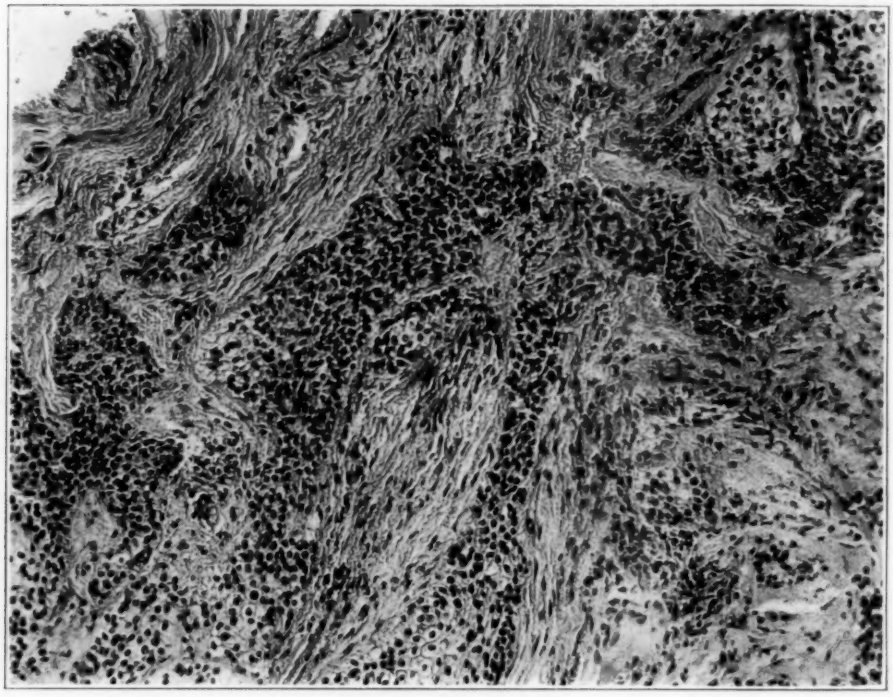

Fig. 2.—High power view of tissue removed from a tracheal neoplasm.

a variable degree of coughing. He had been treated at a large medical center, also privately in New York City and in his home town for "heart trouble." Notwithstanding, he had become gradually worse, to the extent that he could not climb a flight of stairs without becoming short of breath. The past history and family history were noncontributory.

Examination.—Physical examination gave essentially negative results except for a moderate degree of hypotension (100 systolic and 60 diastolic) and the following changes in the chest: On inspiration a curious respiratory wheeze was audible. There was a cough that was suggestively "brassy." The percussion note was everywhere resonant. Auscultation revealed decreased breath sounds over the upper lobe of the left lung and a prolongation of the expiratory phase with very slight harshness. The Wassermann reaction of the blood was negative.

Roentgenography showed the heart and lungs to be normal at the time of admission, and also on repeated occasions throughout the following year. Esophagoscopy proved that the esophagus was uninvolved. Bronchoscopy [6a] revealed a hard, smooth, readily bleeding, sessile mass situated for the most part on the left posterolateral wall of the trachea at a point just above the bifurcation. It occluded about one half of the tracheal lumen.

A specimen was taken for biopsy on Nov. 1, 1930, the tissue being examined by Dr. Arnold Rich, who reported as follows: "Medullary carcinoma, cells growing in cords and sheets suggesting squamous or transitional cell carcinoma."

Treatment and Course.—The growth was fulgurated at its base on November 1. On the second day after operation, there were considerable dyspnea and cyanosis lasting several hours, but they disappeared after the patient had coughed up a mucous plug. Three days later the tumor was once more fulgurated, and again on the second day after operation there were several hours of severe coughing and cyanosis which disappeared after the expulsion of a mucous plug. On the third day after operation, the patient exhibited the classic symptoms of massive collapse of the left lung. This condition was relieved by rolling him back and forth on the affected side for five minutes and then on the normal side for five minutes until he had coughed up a mucous plug, whereupon his respiratory embarrassment soon disappeared. Additional fulguration was done on November 13 and again on November 20, and in each instance the postoperative period was uneventful. The growth was subjected to deep roentgen therapy. A skin dose through four portals to the growth, two portals front and two portals back, was administered at a distance of 50 cm., 200 kilovolts, 30 milliamperes, being used for twenty minutes to each area. Thus he received, during the first month, what was considered to be approximately an erythema dose in the growth itself.

One month after treatment was instituted the tumor was found to have decreased to about one third its original size; moreover, there was no further dyspnea. Two months later, bronchoscopic examination showed the growth to be but one third as large as it was at the start; the patient was entirely symptom-free and had returned to his regular occupation as carpenter. At this time the mass was again fulgurated, the work being performed in the outpatient department, and the patient was able to leave for his home a few minutes after the completion of the operation. Three months later the growth was found to have decreased further in size. It was again fulgurated in the outpatient department. The patient was still symptom-free and had gained 25 pounds (11.3 Kg.). A roentgenogram of the chest at this time failed to show any evidences of metastasis. The patient was seen five months later, and the growth was found to be no larger. It was again fulgurated in the outpatient department. He was free from symptoms and "feeling better than ever." Twenty months later he still remained well.

COMMENT

The foregoing case is noteworthy for the following reasons:

1. Nowhere in the literature have I found any mention of primary carcinoma of the lower third of the trachea that has been as successfully treated simply by a series of electrofulgurations administered by oral tracheoscopy and supplemented by a limited amount of deep roentgen treatment.

6a. The endoscopic work was done by Dr. Edwin F. Broyles.

2. There is on record but one other case in which the results obtained were quite as satisfactory. It is now sixteen months since the patient was first seen, and at present he is in excellent physical condition and has gained 25 pounds. He was able to resume his occupation as carpenter a few weeks after treatment was started. The other case to which I refer is one reported by Figi [7] at the Mayo Clinic. Figi's patient remained well after thirteen months. In his case, however, a tracheotomy was first done before making use of surgical diathermy, and radium was subsequently applied externally.

3. From a prognostic point of view the results obtained are doubly interesting when one recalls the fact that the literature is replete with such statements as "the prognosis in carcinoma of the lower portion of the trachea is practically hopeless," and "patients who have remained well for more than a year following treatment are extremely rare."

4. Tracheal tumors may be confused with early cardiac disease. In fact, my patient, during a period of fifteen months preceding admission, was seen by four different practitioners, and also by a "heart specialist." The latter prescribed a diet, while of the practitioners, two treated him for "heart trouble" and one for "underweight." He was also seen in one of the largest clinics in this country, where the impression was "arteriosclerotic heart disease and moderate decompensation." While I am on the subject of diagnosis, it might be well to mention the following conditions that have been mistaken for tracheal tumors: (*a*) Asthma. An absence of eosinophilia and of eosinophils in the sputum and also failure to improve with epinephrine should put one on one's guard against this mistake. Adam [8] reported three cases of tracheal tumors in which seemingly healthy patients with intermittent dyspnea were erroneously said to have asthma. (*b*) Inflammatory pulmonary diseases. These can usually be excluded by roentgenography. (*c*) Mediastinal tumors. Roentgenography and endoscopy will frequently rule out these conditions.

The literature on primary malignant conditions of the lower third of the trachea is completely covered by D'Aunoy and Zoeller,[2] Broman, Fraenkel, Holmgren, Maier and Thiesen for the period extending through 1929. Since then I have found but five other cases reported. Neilson [9] mentioned the case of a man of 54, who, at autopsy, was found to have a primary malignant process located about 2.5 cm. above the bifurcation. The esophagus was also slightly involved. The patient

7. Figi, F. A.: Primary Carcinoma of the Trachea, Arch. Otolaryng. **12**:446 (Oct.) 1930.

8. Adam, J.: Four Cases of Tracheal Tumor, J. Laryng. & Otol. **41**:174, 1926.

9. Neilson, D. F. A.: Case of Primary Carcinoma of the Trachea, J. Laryng. & Otol. **45**:855 (Dec.) 1930.

died before treatment could be instituted. Breslich [8] reported the case of a man, aged 60, with a primary carcinoma 2 cm. above the bifurcation, who died two days after he was first seen. No treatment was attempted. Figi [7] of the Mayo Clinic mentioned two cases, in both of which the tumor was situated low down in the trachea. One patient received roentgenologic treatment, but succumbed three months later. The other patient was not treated and died seven weeks after the diagnosis was made. Gaillard [10] and Barmwater [11] each mentioned a case of primary malignant tumor of the trachea, but I am not certain that the tumors to which they referred were located in the lower third of the organ. Chevallier [12] reported the case of a man of 55, who, at autopsy, was found to have primary epithelioma at the bifurcation. The patient died five days after he was first examined having received no treatment other than a tracheotomy.

SUMMARY

1. Primary malignant tumors of the lower third of the trachea are extremely rare.

2. Few cases are recognized until so far advanced that treatment is of little, if any, avail.

3. The symptom complex of intermittent dyspnea, cough, perhaps occasional hemoptysis, apparent good health and absence of obvious intrathoracic signs suggests tumor in the tracheobronchial tree. In such cases endoscopy is most certainly indicated.

4. A review of the literature up to July 1, 1931, was made.

5. A case of primary malignant tumor of the lower third of the trachea is presented.

6. The results obtained in the treatment of a patient with a primary malignant condition of the lower third of the trachea by the use of intratracheal electrofulguration, supplemented by deep roentgen therapy, are most encouraging and merit further trial.

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10. Gaillard, R.: Primary Epithlioma of the Trachea: Case, Rev. de laryng. **51**:691 (Dec. 15) 1930.

11. Barmwater, K.: Primary Cancer of the Trachea, Hospitalstid. (Dansk otolaryng. selsk. forh.) **73**:44 (Oct. 9) 1930.

12. Chevallier, R.: Primary Carcinoma of the Trachea Beginning with Dysphagia: Case, Lyon méd. **146**:309 (Sept. 14) 1930.

EXTRACTION OF FOREIGN BODIES FROM THE FOOD AND AIR PASSAGES BY DIRECT METHODS

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The inclusion of the tracheobronchial tree and esophagus in the specialty of laryngology dates back thirty years. While the exploration of the former is due entirely to the genius of Gustav Killian and is considered as new territory, the work on the latter is principally due to the efforts of von Mikulicz, although such pioneer laryngologists as Semeleder, Stoerk and others have also contributed a great deal to the subject.

Tracheobronchology must be looked on as a direct continuation of laryngology and is indispensable to the laryngologist. The laryngologist's relation to the esophagus, however, is the result of manifold connections in the pathology of the contiguous food and air passages, which must be considered in the differential diagnosis of conditions in both organs.

Peroral endoscopy claims a complicated technical apparatus and a careful methodical education.

Because the opportunity of application and practice of endoscopic methods is relatively rare and, on the other hand, their indications have a more direct vital importance, it is necessary to possess a thorough training before one attempts to do this highly specialized work.

While work in this special field is not plentiful and only seldom remunerative, it is at least fascinating and at times dramatic, especially when one meets with a foreign body in the depths of the bronchial tree.

The removal of foreign bodies from the esophagus may be less spectacular, but it is certainly much more difficult and dangerous. It is true that every intern can pass a stomach tube, but only the most efficient students of endoscopy should attempt to pass an esophagoscope. In the aged, the walls of the esophagus are friable, and tears or perforations may result in the hands of the most expert surgeon.

In infants and very young children, because of the narrow lumen and tender tissues, much damage has been done by the best laryngologists, and at times the damage is beyond repair. However, this cannot be helped, and while experts are necessary to do this work, all laryngologists cannot become expert at once.

CHOICE OF ANESTHESIA

For adults in all forms of endoscopy, I use local anesthesia in the form of 10 per cent cocaine hydrochloride to which is added a few drops of epinephrine chloride 1:1,000. For children no anesthesia is given. Of course, there are children, especially between the ages of 7 and 14, for whom a general anesthetic becomes imperative. Such children are usually unmanageable and difficult to handle in any other way.

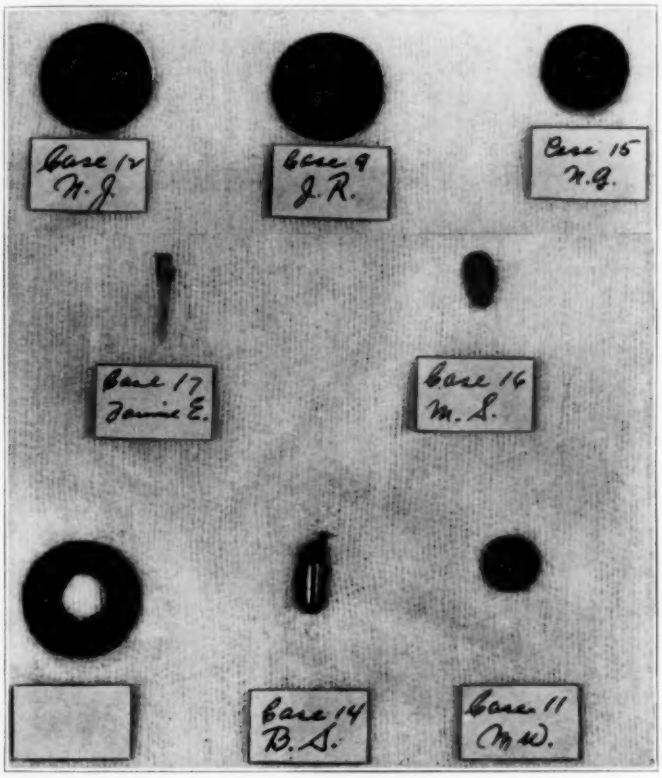

Fig. 1.—Foreign bodies removed in several cases.

REPORT OF CASES

CASE 1.—*History.*—P. R., a man, aged 52, was referred to me on Jan. 17, 1926, by Dr. Hyman Glanz, with a complaint of inability to swallow food and drink and a burning and choking sensation in the throat for the past five days. These symptoms came on suddenly following his evening meal on January 12. He vomited on several occasions and coughed a good deal without raising any expectoration.

He saw several physicians, who took roentgenograms, performed fluoroscopy, pumped and washed his stomach and assured him that nothing could be found and

nothing was wrong. With the foregoing history he came to me in great distress and looking acutely ill. He was so frail from starvation and repeated examinations that he could hardly walk.

Physical Examination and Treatment.—Nothing abnormal was found in the fauces, pharynx and larynx. On exploring the esophagus with olive-tipped flexible metal bougies, an obstruction was found about 8½ inches (21.5 cm.) from the teeth which was very painful; only the smallest olive could be forced through the obstruction.

In order to determine the character of the obstruction, I passed, under local anesthesia, a short esophagoscope, Seiffert type, and as it forced its way through the obstruction I suddenly observed a welling up of pus from the obstructed area. The pus was dirty gray and had an offensive odor. An abscess in this locality made me think of a foreign body, and as the Seiffert esophagoscope was too short for deeper exploration and nothing else could be seen except an abscess cavity, I decided to use a longer esophagoscope.

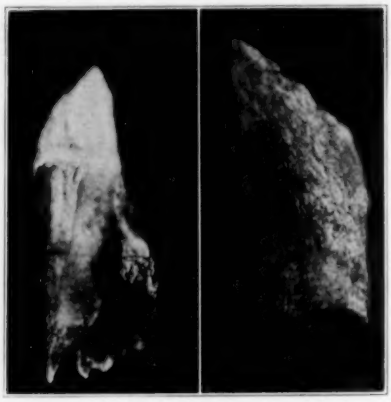

Fig. 2 (case 1).—Two views of the meat bone in the abscess cavity just beyond the mouth of the esophagus.

With the patient in the recumbent position, I passed a medium-sized Mosher oval esophagoscope and just at the bottom of the abscess cavity observed a sharp bone, which I grasped with forceps and removed.

The foreign body turned out to be a good-sized meat bone 1½ inches (3.77 cm.) in length and three fourths of an inch (1.87 cm.) in width, one extremity of which was pointed and the other broad and sharp. The pointed extremity was turned upward in the wound, and it was at this extremity that the seizing forceps was attached and the bone removed.

Swallowing movements, while still somewhat painful owing to the open wound of the abscess cavity, were no longer unbearable. Three days later the patient was discharged, having made an uneventful recovery.

Case 2.—J. T., an Italian boy, aged 11 years, was admitted to the Beth David Hospital, in the service of Dr. Lowinthan, on Jan. 29, 1926. His father had presented him with a quarter for being good in school; he put it in his mouth and accidentally swallowed it. He was taken to the hospital, complaining of a pressure sensation deep down in his throat with but slight interference in swallowing food. He suffered from no pain and did not look at all anxious.

A roentgenogram was promptly taken, and a shadow the shape of a coin was located in front of the vertebral column midway between the fourth and fifth ribs.

The patient was given 1/10 grain (0.006 Gm.) of apomorphine by hypoderm to induce vomiting, which he did, but this did not dislodge the coin. Esophagoscopy was then advised.

This procedure was carried out on January 30. Before proceeding with the actual esophagoscopy, I attempted to remove the coin with an old-fashioned coin catcher, but unfortunately I failed in the attempt, and the only thing I gained was to injure the posterior wall of the mouth of the esophagus.

Direct esophagoscopy was then carried out under general anesthesia, the small Bruening esophagoscope being used. A coin with a serrated margin was sighted at about the second esophageal constriction and easily removed with a long seizing forceps. The boy made an uneventful recovery and left the hospital three days later.

CASE 3.—John White, aged 42, was admitted to the Beth David Hospital on June 27, 1926. While eating his evening meal the night before he swallowed a fish bone, which stuck in the throat and gave him painful and difficult swallowing.

After a searching examination of the root of the tongue, pharynx, hypopharynx and piriform sinuses revealed nothing, a medium-sized, oval esophagoscope (Mosher type) was passed. The mouth of the esophagus was found abnormally closed, and much time and patience were spent in gradually dilating this orifice. After a prolonged search, a fish bone was found completely buried in the left lateral wall at the first constriction of the esophagus. The bone had to be milked out with the tip of the esophagoscope so as to make it accessible to the grasping forceps. It was found to be 2 inches (5 cm.) long, rigid and, at the distal end, pointed and sharp. The patient left the hospital the following day entirely well.

CASE 4.—Infant Edelstein, aged 1 year, was admitted to the Beth David Hospital on June 16, 1925, in the service of Dr. Maximilian Schulman. The condition began suddenly, with spasmodic coughing, wheezy breathing, dyspnea and cyanosis. The physical signs also pointed toward a foreign body in the right lung. A roentgenogram of the chest, however, revealed no foreign body.

Bronchoscopy, under ether anesthesia, with the smallest Bruening bronchoscope revealed no foreign body in either lung, nor any other lesion in the lower air passages. Examination of the interior of the larynx revealed marked swelling of the mucous membrane of the ventricle, the false cords and the subglottic space. The swelling was no doubt responsible for the symptoms, as the child was sent home the next day and soon made an uneventful recovery. An oily solution of epinephrine dropped into the nose several times a day was the only treatment.

CASE 5.—Ervin Schiff, aged 2 years, was referred to me on July 30, 1926, by Dr. Goodheart, and admitted to the Jewish Memorial Hospital. Five days before admission, the child was playing with a nickel by tossing it up in the air while his mouth was wide open. The coin fell into the mouth, was swallowed and forced into the esophagus. With the smallest Bruening esophagoscope, under ether anesthesia, the nickel was easily removed from the diaphragmatic constriction of the esophagus. The child made an uneventful recovery and was discharged the following day.

CASE 6.—Baby Ford, aged 1 year, was seen on July 4, 1927, in consultation with Dr. Solomon Handleman. One week prior to consultation, the child had swallowed a collar button. Without anesthesia, a short infant-sized esophagoscope was inserted, and the collar button was found hemmed in the wall of the esophagus at the second constriction.

An attempt was made to turn the collar button so as to dislodge it from its hold in the walls, without success. As this was not possible, we tried to move the invader by simple traction on the neck of the button. By doing so the walls of the esophagus gave way, and we could distinctly see the trachea through the perforation. The operation was abruptly terminated and the child rushed to the hospital.

A roentgenogram was taken, and the collar button was located in the cervical esophagus.

As the child was too sick to be meddled with further and the neck and chest were emphysematous, he was put to bed. Mediastinitis set in, with death in forty-eight hours.

Case 7.—E. G., a boy, aged 3½ years, was referred by Dr. Mark Reuben, and was admitted to Beth Israel Hospital on Oct. 16, 1927. Twenty-four hours before admission the child had swallowed a nickel and had since vomited several times and complained of substernal pain. Roentgen examination showed a coin in the lower cervical region. The vertical axis was to the left of the midline.

Esophagoscopy, without anesthesia, was performed on October 17. The coin was removed, and the child was discharged the following day in good condition.

Case 8.—Mrs. S. J., aged 65, was referred to me by Dr. Adolph Greenstein on Nov. 15, 1927. While eating she began to choke and felt the pressure of a foreign body in the throat. She tried to dislodge the foreign body by swallowing bread and potatoes, without success. Later the patient felt a sticking pain in the throat.

Esophagoscopy was performed under local anesthesia; 10 per cent cocaine to which a few drops of epinephrine were added was used. With a Seiffert sideways opening esophagoscope a chicken bone about 2 inches (5 cm.) long covered with meat was located 3 inches (7.6 cm.) beyond the mouth of the esophagus. The bone was removed with a Jackson esophageal forceps. The patient made an uneventful recovery and left the hospital the following day.

Case 9.—J. R., a boy, aged 5, years, was admitted to the United Israel-Zion Hospital on June 28, 1928, with a history that he had swallowed a quarter on June 19. Since then he had complained of substernal pain and had been able to eat only with difficulty.

Esophagoscopy was performed on June 29, without anesthesia; the coin was found wedged in the diaphragmatic constriction of the esophagus. It was removed with an alligator forceps. The coin was a quarter very much corroded, evidently from its long sojourn in the esophagus close to the stomach. The patient made an uneventful recovery and was sent home in a few days.

Case 10.—N. C., aged 19 months, was admitted to the United Israel-Zion Hospital, on Nov. 10, 1928, with a history of having swallowed a metal clasp from a garter on November 8.

Esophagoscopy, without anesthesia, was performed on November 11; it revealed a metal clasp located somewhat beyond the second constriction of the esophagus. Its long diameter was parallel with the long axis. An attempt to remove the clasp with grasping forceps failed, and only pushed the clasp further down the esophagus. After several attempts the clasp disappeared from view. A roentgenogram taken at the time revealed the foreign body in the stomach from where the child passed it out twenty-four hours later.

Case 11.—M. W., aged 11 years, was referred to me by Dr. Jacobson, with a history of having swallowed a thumb tack on the morning of June 29, 1928.

Esophagoscopy was performed at Beth David Hospital without anesthesia, a Seiffert short esophagoscope being used. The tack was located at the second

constriction with its pointed edge stuck in the right wall of the esophagus. The tack was easily removed with a grasping forceps by first taking it out of the lateral wall and engaging its tip into the mouth of the grasping forceps, which was removed together with the esophagoscope. The child was sent home in good condition the next day.

Fig. 3 (case 9).—Coin in the upper part of the esophagus, which was found on esophagoscopy hemmed in the diaphragmatic constriction.

Fig. 4 (case 11).—Two views of the tack, located in the cervical esophagus.

CASE 12.—N. J., a girl, aged 10 years, was referred by Dr. Mark Reuben. She was admitted to the Beth Israel Hospital on June 30, 1928, with a history of having swallowed a quarter two days prior to admission.

A roentgenogram located the coin in the cervical esophagus. It was removed by direct esophagoscopy under general anesthesia on the day of admission. The child was discharged from the hospital on the following day in perfect health.

CASE 13.—Irving C., aged 7 months, came to my office on July 14, 1928, with a history of having swallowed a safety-pin. The mother had an x-ray picture

that showed an open safety-pin, sharp end downward, in the front of the upper cervical vertebra.

Laryngoscopy, without anesthesia, revealed an open safety-pin the lock end of which was resting in the larynx and the sharp pointed end of which was buried in the wall of the right piriform sinus astride the right aryepiglottic fold. The pin was grasped and easily removed with a Jackson laryngeal forceps. Recovery was uneventful.

Case 14.—B. S., a boy, aged 5 years, was referred to Beth David Hospital, in the service of Dr. Sobel, on Nov. 20, 1928. The diagnosis on admission was traumatic pneumothorax. A roentgenogram, however, revealed a foreign body in the left lung.

Bronchoscopy was performed on the following day without anesthesia, a child-sized Bruening bronchoscope being used; a smooth metallic foreign substance was

Fig 5 (case 13).—Safety-pin in the larynx, point downward, stuck in at the piriform sinus.

located in the left main bronchus. This was removed with a Bruening fenestrated forceps and proved to be a small bulb of an electric otoscope.

This case was described in detail elsewhere by both Dr. Sobel and myself.

Case 15.—N. G., a girl, aged 2 years, was admitted to the United Israel-Zion Hospital on Jan. 1, 1929, with a history of having swallowed a one cent piece twelve days before admission. The complaint on admission was difficulty in swallowing immediately after feeding.

Roentgen examination revealed an opaque metallic coin at the cervicothoracic junction. With a Seiffert esophagoscope, without anesthesia, the coin was easily grasped and removed. It was found to be a much corroded one cent piece.

Case 16.—Marion S., aged 10 months, was referred to me by Dr. A. Noah Schiller on July 1, 1929, with the following history: On June 28, while the child played on the floor, it picked up a foreign substance, put it in its mouth, choked on it, turned blue and began to cough. The mother at once turned the child over

and tried to shake the invading substance out. As this proved unsuccessful, she took the child to her family physician, who at once sent it to the Vassar Hospital in Poughkeepsie.

There roentgenograms were taken, and the roentgenologist reported a suspicion of a foreign body in the lower air passages. A bronchoscopy was advised, and the child was brought to New York and admitted to the Jewish Memorial Hospital in the service of Dr. A. Noah Schiller.

Roentgenograms taken at the Jewish Memorial Hospital revealed no foreign body anywhere in the lower air passages. Physical examination of the child at this time revealed no symptoms suggestive of a foreign body in the tracheobronchial passages. The child looked pink and had no difficulty in breathing, no cough and no elevation in temperature. The mother, however, insisted that the child had aspirated a foreign body and wanted the same removed.

Bronchoscopy was performed on July 1, four days following the alleged aspiration of the foreign body.

Without anesthesia, the child was completely mummified, and a Killian tube, size 6 mm., was attached to the Bruening electroscope and inserted through the glottis into the trachea down to the carina; a foreign body was found at the entrance to the right main bronchus. This foreign body was placed horizontally so that air could pass easily above and below it. With a fine grasping forceps, Jackson type, the foreign body was grasped and removed. It proved to be a watermelon seed. The child made an uneventful recovery.

CASE 17.—Fanny E., aged 43, was referred to me by Dr. A. M. Schwager on Nov. 14, 1929. Her history dated back to two days prior to her visit to my office, when she had eaten a piece of fish and thought that she might have swallowed a bone. Since then there had been marked pain on swallowing, especially on the left side, which became progressively worse and at the time she entered my office was unbearable. She was admitted to the Beth Israel Hospital, and on the same day esophagoscopy was performed under local anesthesia, a short Jackson esophagoscope being used.

A fish bone was located at the left lateral wall of the esophagus about an inch below its mouth. The surrounding area was markedly congested and edematous; the fish bone was deeply buried for more than three fourths of its length. The free end of the fish bone was only about 1 cm. in length and was easily removed with a Jackson alligator forceps. Following removal the agonizing pain disappeared, but the patient still had difficulty in swallowing for the next twenty-four hours. She was then discharged free from all pain and symptoms.

CONCLUSIONS AND COMMENT

It is important to remember that esophagoscopy is much more difficult and dangerous to perform than bronchoscopy, especially in infants and the aged.

Local anesthesia is used for adults; neither general nor local anesthesia is used for children, with few exceptions.

Patients with upper dental plates have difficulty in detecting foreign bodies, especially bones, etc., and easily swallow them.

Occasionally some patients give classic signs and symptoms of a foreign body in the air passages which may be due to other causes, such as subglottic edema.

In perforations of the cervical esophagus, when one is sure of the perforation, it is advisable to do a prophylactic mediastinotomy through the neck, Marschek's method. Perforations lower down in the esophagus should be enlarged, and the patient should be fed via a rubber tube inserted through the nose, thus draining the mediastinum, Seiffert's method. The use of the old-fashioned coin catcher should be condemned.

At times a foreign body is dislodged from the constricted area by forceps or a scope and is pushed downward into the stomach.

305 East Seventeenth Street.

Clinical Notes

INTRANASAL OPENING OF THE MAXILLARY ANTRUM BY ELECTROCUTTING AND COAGULATION

Louis Hubert, M.D., New York
Assistant Surgeon, Manhattan Eye, Ear and Throat Hospital

In many articles on electrosurgery attention is called to the danger of necrosis of the bone if during the operative procedure the active electrode happens to come in contact with periosteum. I was aware of that risk when I started this work, a little over a year ago, in Dr. Francis White's clinic of the Manhattan Eye, Ear and Throat Hospital. I proceeded with great caution and soon found that osseous necrosis of the maxillary antrum can be avoided by a proper technic and by a proper selection of patients.

TECHNIC

At the Manhattan Eye, Ear and Throat Hospital my associates and I use a low voltage high frequency machine. It is probable that a high voltage machine is not suitable for electrosurgery on the antrum, because the higher the voltage the more readily will sparks occur at the cutting electrode, and such sparks tend to carbonize the margins of the wound and may thus cause necrosis of bone. Of utmost importance in this work is the proper setting of the machine. For electrosurgery on the antrum the controls of the apparatus are so set that a current of 3,000 milliamperes is obtained if the two binding posts are short-circuited by a piece of wire. With such a setting a large indifferent electrode of from about 60 to 70 square inches is employed in children, who are operated on under general anesthesia, or a spring jawed arm electrode is utilized in adults, in whom local anesthesia is always used. The local anesthesia must be as thorough as when a regular intranasal antrum operation is performed. If the anesthesia is not complete, the patients become nervous on account of a peculiar reflex pain in the teeth and resist further manipulation.

The proper handling of the active electrode, which is an insulated trocar (see illustration), is of great importance. This electrode, which is connected to one of the binding posts of the machine, is placed underneath the inferior turbinate at about the point at which a trocar for washing out the antrum is usually placed. When the machine is properly set and operated with a foot switch, the electrified and insulated trocar may within a few seconds cut its way through into the antrum, if the naso-antral wall is very thin; or, with the current still on, the trocar is pushed through with a moderate amount of force, if the wall is thicker. As the trocar enters the antrum the insulating rubber is partly shoved back during the procedure, and a larger area is thus exposed for coagulation. The current is then interrupted

Read before the Section of Otolaryngology, New York Academy of Medicine, May 18, 1932.

by the foot switch, and the trocar is removed. It is surprising to find an opening in the antral wall from four to six times the diameter of the trocar used. In thick walls the opening may not be large enough, and the trocar is then reintroduced through the same opening and the tissues around the opening are coagulated again. Care must be taken not to make a new opening in the naso-antral wall, as necrosis of bone between the two openings may follow. Care must also be taken that the trocar does not touch the interior walls of the antrum, as sloughing and severe reactions may follow. Cautious handling of the trocar is therefore essential for the success of this procedure.

Selection of Patients.—The first patients who were submitted to the procedure described had undergone operations on the antrums by the usual methods, but the antral openings were practically closed when reexamined some time after the primary operation. When the electrified trocar was placed in the space where the original opening was made, it cut through the scar tissue and granulations, and a large opening with no tendency to close remained. This method was then tried on patients with chronic antral disease who had not been operated on before. There was no appreciable reaction in patients with moderately thin antral walls. In patients with very thick naso-antral walls a great deal of force had to be used in order to break through the walls. A moderate reaction usually followed this pro-

Electric trocar.

cedure. During the epidemic of influenza in 1932 I used this method in two cases of acute antral disease, and a severe reaction followed in both cases. It should therefore never be used in acute conditions of the antrum and with great care in chronic conditions when the antral walls are very thick. It is an ideal method for patients whose antrums have been operated on before, but in whom the openings are more or less closed, and in chronic cases when the naso-antral walls are thin.

With due precautions and careful selection of patients, the results of the procedure described in this paper are very good. The openings made in the naso-antral walls are large and show little tendency to close even without after-treatment. For several weeks bare bone is detectable around the openings. Although no necrosis of bone can be made out, a slight degree of it, just enough to keep the openings patent, probably occurs. After a time the bare bone becomes covered with mucous membrane.

What are the advantages of this operation? If carefully performed, it is very simple, requires only a few seconds and surpasses in its results the more elaborate operations usually performed for the same purpose. There is little or no bleeding during the operation and no postoperative hemorrhage. Adhesions, which are frequently formed between the inferior turbinate and the septum after the usual operations, are conspicuous by their absence. Very little after-treatment is necessary. This operation is therefore especially of service in children in whom an operation on the antrum is indicated.

If the various precautions indicated in this paper are carefully observed, there are practically no disadvantages. However, this operation cannot be used indiscriminately, and it is likely to do a great deal of damage if not properly performed.

In conclusion, I believe that the operation described represents progress in intranasal operations on the antrum.

161 East Seventy-Ninth Street.

NASAL POLYPI: TREATMENT AND PREVENTION OF RECURRENCE

J. Coleman Scal, M.D., New York

Nasal polypi are tumors originating from the mucous membrane of the nasal cavity. These polypi are true hypertrophies of the mucous membrane of the parts involved and possess the epithelium of their source. The origin of nasal polypi is still undetermined.

Zuckerkandl believes that nasal polypi are the result of an inflammatory process of the nasal mucous membranes. He draws his conclusions from the fact that he has found round cell infiltration in large amounts and free cells in the exudate in even mild cases.

According to Freedman,[1] nasal polypi are not new growths, but are a localized edema and prolapse of the nasal mucous membrane, resulting either from infection or from trauma. He believes the polyp to be a symptom of an underlying osteomyelitis or periostitis of the sinus, and substantiates his theory by stating that removal of the nasal polyp will not result in a cure unless attention is given to the underlying cause.

According to Hajek,[2] polypi are edematous hypertrophies of the mucous membrane of the nose. He believes that in exceptional cases they can begin in the bone of the invaded area. He came to this conclusion by noticing a case in which a middle turbinate had been removed and which had developed on the severed surface an edematous swelling similar to a polyp. Examination of this showed edematous connective tissue which originated from the prolapsing of the marrow spaces which were opened up in the original operation.

Cordes confirmed Hajek's findings, but he claimed that simple nasal polypi have their source in the nasal mucous membrane, but that recurring polypi originate as the result of bone changes, such as osteitis.

According to Grunwald,[3] there is an etiologic relationship between polyp formation and suppurative sinusitis, the polyp formation being produced secondarily by chronic suppuration.

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1. Freedman, A. O.: The Pathogenesis of Polyposis Nasi, Arch. Otolaryng. **2**:250 (Sept.) 1925.

2. Hajek, M.: Pathologie und Therapie der entzündlichen Erkrankungen der Nebenschen der Nase, Leipzig, 1926; translated and edited by Joseph D. Heitzer and F. K. Hansel, St. Louis, C. V. Mosby Company, 1926.

3. Grunwald, Ludwig: Nasal Suppuration of the Nose and Its Accessory Sinuses, translated from the 2d German Edition by William Lamb, New York, William Wood & Company, 1900.

The recurrence of nasal polypi has been explained in several ways by several authors. Zuckerkandl stated that it is due to incomplete removal. Hajek and Alexander claimed that recurrence is not a regrowth, but new formation of polypi as the result of deep invasion of the inflammatory process to the bone marrow, and that this stimulates the mucous membrane to new polyp formation. According to Grunwald, recurrence is due to neglected sinus empyema, and in some cases to a polypoid degeneration of the mucous membrane of the accessory sinuses.

Nasal polypi are found most frequently in the ethmoid region and less frequently in the maxillary, sphenoidal, ethmoidal and frontal sinus regions. Some polypi originate from the middle turbinate bone, but these show no tendency to recur after removal.

The therapeutic methods employed to date have been unsatisfactory. Operative procedure will relieve the symptoms, but if the original cause be not eradicated the polypi will form again in from one to three years. The removal of nasal polypi by the use of radium element has been attempted but has been discarded as ineffective.

The best surgical procedure for the elimination of nasal polypi, at the present time, is the removal of the polypi and the eradication of any existing pathologic nasal condition. Immediately following this, radium element should be applied to the site of the polyp.

The patient's nares are cocainized by inserting cotton pledgets saturated with equal parts of 10 per cent cocaine and epinephrine. After a ten minute application the cotton is removed and 2 per cent procaine hydrochloride and epinephrine is injected into the ethmoid region, especially behind the middle turbinate. The polypi are then engaged with a Krause snare and removed. Those inaccessible to the snare should be removed by the forceps. Bleeding is almost nil, but if it is encountered it can be checked readily by narrow strips of hemostatic gauze inserted into the nares. When the ethmoids are involved an exenteration of the ethmoidal cells with or without sacrificing the middle turbinate bone is the proper procedure. If the maxillary sinus is involved, a wide opening made in the lateral nasal wall is sufficient for drainage.

After this operation several days are allowed to elapse until the reaction, if any, has cleared up. Then a 50 mg. capsule of radium element, screened by 1 mm. of platinum in the form of a container which in turn is screened by guttapercha, is placed into the nostril against the site of the origin of the polypi. It is supported high up in the nostril by a tapering cotton tampon and is held securely by a wooden applicator as well as by a strong black thread. This makes it impossible for the radium to slip into the nasopharynx and be swallowed. The radium is permitted to remain in situ from two and one-half to three hours, giving a dose of from 125 to 300 millicurie hours.

Two such applications are administered one week apart. No reaction following the radium application has been noted, except in a few cases in which the complaint has been that of an acute cold for twenty-four hours.

CONCLUSION

Seven years' experience in this therapy has convinced me that it should be used after the removal of polypi in order to prevent a recurrence of this annoying condition. I find that in over a hundred cases in which this treatment was used only three patients returned with a regrowth of polypi. One had a recurrence one year after removal and the other two years after operation. In each case, however, the polypi were small and they were not of the boggy, edematous, oyster type but seemed to be of a more fibroid character, quite small and easily removed.

In conclusion, I should add that in most of these cases of nasal polypi the tissues and structures in the supranasal fossae seem to be congested after operation, often making the entrance of the radium difficult. Several weeks after the application of the radium I find this opening more roomy, permitting freer aeration of this chamber. No deleterious effect has resulted in a single case in which this manner of treatment was used.

44 West 74th Street.

A NEW RHINOSCOPE

OSCAR SWINEFORD, JR., M.D., UNIVERSITY, VA.

Inspection of the nasal cavity is generally omitted in routine physical examinations, in spite of the fact that nasal disease is responsible for many of the symptoms for which patients consult physicians.

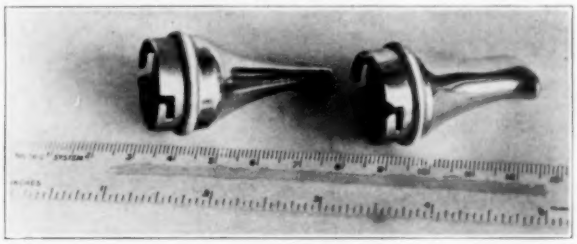

Fig. 1.—Nasal speculum (right) adapted for use with otoscope frame and light. The standard ear speculum is shown for comparison.

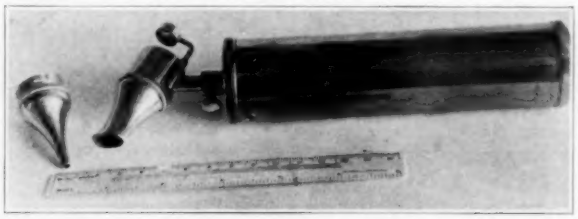

Fig. 2.—Rhinoscope. Nasal speculum fitted to otoscope frame and light. The standard ear speculum is shown for comparison.

For the past few years my associates and I have urged our students in physical diagnosis and in internal medicine to describe the condition of the structures in the nasal cavity of every patient they examine. This was done by using the large speculum of their otoscope sets. This arrangement, however, did not permit inspection of the whole cavity at once.

It occurred to me that a nasal speculum fitted to the otoscope frame and light would permit adequate inspection. Accordingly arrangements were made with the American Cystoscope Makers, Inc., to adapt a Simrock no. 2 nasal speculum to one

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From the Department of Internal Medicine, University of Virginia Medical School.

of their Wappler otoscopes. The result has been very satisfactory. With it (fig. 2) the nasal structures can be seen just as well as with the nasal speculum and head mirror. In the wards and in homes it obviates the necessity for portable drop lights which are required when head mirrors are depended on for the illumination of the nasal cavity. Topical applications can be made with facility.

We have been using the combination otoscope-ophthalmoscope sets as a routine to transilluminate sinuses, to examine retinal fields, ears and throats and to test pupillary reactions to light. It is felt that this new speculum will greatly increase the usefulness of the set. With it the oto-ophthalmoscope becomes an otorhino-ophthalmoscope.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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INTRADURAL CONDITIONS IN RELATION TO RHINOLOGY AND OTOLOGY

A CRITICAL SURVEY OF RECENT LITERATURE

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

INTRODUCTION

During the past few years America has produced a number of research workers, Shambaugh, Blast, Crow, McMahon, Hassin and Martland, whose microscopic studies, like those of Frazer, Turner and Reynolds in England, have added greatly to our knowledge of normal processes as influenced by infections. They are carrying on and enlarging the work begun by Schwartze, Gruber, Seibenmann, Politzer and Alexander.

Development of Language.—In "Babel, or Language, Past, Present and Future," [1] Paget suggested that all human language originated from gestures and all written language from signs similar to the original American Indian sign language. Gestures are accompanied by sounds made by associated movement of the tongue and lip. These primary sounds are the elements of all vocal language, that is, a combination of sound with gesture. Paget stated that immediately following the Norman Conquest, the language used by the conquerors consisted of Greek and Latin, from which came all the Romance tongues. Latin was used by the clergy, and French by the nobility. But the Anglo-Saxons still used their primitive gesture or sound language based on the emotional movement of the lips and tongue. Because of the fundamental emotional, gestural and muscular basis of the Anglo-Saxon tongue, these sounds were gradually absorbed into the Latin and French. This is another case in which a primitive people, although conquered, overcame by the force of its innate truths a higher and more artificial civilization. Paget called attention to the fact that all the prefixes in

1. Paget, Richard: Babel, or Language, Past, Present and Future, London, George Routledge & Sons, Ltd., and Kegan Paul, French, Trubner & Co., Ltd., 1930.

English are made from sounds many of which the animals can almost produce. The sounds have been modified by the development of the human larynx and the tongue.

Cerebral Centers for the Location of Sound.—Greene [2] found that a study of the hearing in persons with tumor of the temporal lobe favors the view that auditory pathways extend from each ear to both temporal lobes. They form a decussation similar to that in the optic chiasm. Clinical evidence favors the view that part of the temporal lobe probably is the region of the brain that contains a center enabling one to localize sound. There probably is a center in each temporal lobe. Possibly there is only one center. The hemisphere in which this center is situated depends on whether the patient is right-handed or left-handed. In case there are two centers, the sensation may be complicated by nerve tracts connecting the two centers, perhaps crossing in the corpus callosum. Greene concluded that there are two possible interpretations of the clinical observations: (1) Lesions of the temporal lobe tend to impair the ability to localize sound, and (2) increased intracranial pressure also tends to impair this ability.

Sensory Music Center.—The results of a macroscopic and microscopic study of the brain of the opera singer, G. Pogany, and the violinist, J. Nagy,[3] can be summarized as follows: 1. In both brains a cerebral cortical development, greater than normal, was present in the same cerebral areas that had been found to be overdeveloped in the brains of other musically talented people which have been studied by Grossman, Auerbach and Klose. These areas are located in the middle portion of the left upper temporal convolution, the lower segments of the left parietal lobe, and more particularly, in the supramarginal gyrus, as well as similar areas of the right hemisphere. In both brains marked development was also present in Broca's convolution. 2. These cases, as well as the literature, suggest that the sensory music center is in the middle third of the left upper temporal convolution with participation of the right side. 3. Somogyi is of the opinion that the morphologic cerebral changes associated with the higher development of the musical faculties consist chiefly in an enlargement of cortical areas. Internal growth is also necessary, a qualitatively or quantitatively supernormal, cytologic and myelogic structure. 4. In Somogyi's opinion, the enormous development of the centers has an endogenous hereditary basis, in which an important part cannot be assigned to increased function, as a factor inducing hypertrophy.

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2. Greene, T. C.: The Ability to Localize Sound, Arch. Surg. **18**:1837 and 1841 (April) 1929.

3. Somogyi, I.: Ueber das morphologische Korrelat der musikalischen Fähigkeiten, Monatschr. f. Psychiat. u. Neurol. **75**:138 (March) 1930.

Colored Audition.—The association of an auditory with a visual sensation is rare in normal adults but more frequent in pathologic cases. It consists in the appearance of visual images, usually geometric forms, following the pronunciation of certain sounds: vowels, consonants, names of persons, days of the week or isolated notes of the violin.

Marinesco and Vasile [4] expressed the belief that this is a psychologic condition. It is a predisposition that is associated with a greater impressionability of the auditory centers for words and of mental vision. Colors associated with vowels, names of countries, and other things are specific for the same person.

Preferential associations play an important rôle. One remembers certain things encountered only once but which are deeply engraved on the mind. Thus, one patient associated Sunday with red because the calendar Sunday dates are printed in red ink. In the majority of patients the color representation is only mental, but a number of patients who are blind project the color vision. Thus, one patient sees the image in front of his face to the right and above his head. In one case, the sensibility was so marked that the patient colored words when she wrote them or read them to herself. When she read mentally, she heard the words as if they were spoken.

INTRACRANIAL NONSUPPURATIVE AUDITORY LESIONS

Auditory Hallucinations.—Berggren [5] reported thirty-six carefully examined cases of auditory hallucinations from the Insane Asylum St. Lars, in Lund. Auditory hallucinations were noted in ten insane persons with normal ears and in twenty-six with ear disease. In four cases with unilateral auditory hallucinations, there was a unilateral ear condition. Berggren expressed the opinion that auditory hallucinations occur in a large percentage of insane persons with ear disease. It is not known what part peripheral diseases of the auditory organ play in the origin of auditory hallucinations. The author's investigations do not demonstrate that the ear disease is a causative factor in auditory hallucinations. In four cases of presbycusis, the patients had hallucinations for several years prior to the onset of the ear disease.

Deafness from Degeneration of the Auditory Central Tract.—Crowe [6] studied the disturbances in hearing and the microscopic changes

4. Marinesco, G., and Vasile, Sava: Studies of Colored Audition, Rev. d'oto-neuro-opht. **7**:500 (July) 1929; abstr., Arch. Neurol. & Psychiat. **23**:818 (April) 1929.

5. Berggren, Sture: Relations Between Auditory Hallucination and the Auditory Organ, Arch. f. Ohren.-, Nasen- u. Kehlkopfh. **120**:141 (March) 1929.

6. Crowe, S. J.: Anatomic Changes in the Labyrinth Secondary to Cerebellopontile and Brain Stem Tumors, Arch. Surg. **18**:982 (April) 1929.

in the ear that are associated with cerebral arteriosclerosis, syphilis, hypertension, hypotension, nephritis, disturbances of the ductless glands, the various diseases of the blood, and lesions of the central nervous system. In case 1, the changes in the inner ear were secondary to a lesion of the cochlear nerve within the internal auditory meatus; in case 2, similar changes were due to a lesion of the auditory pathway within the cerebellum and midbrain. These cases would suggest that the wallerian law of degeneration does not apply to the ganglion of the cochlear nerve.

The cochlear nerve, according to the generally accepted view, is like a sensory spinal cord nerve in that a lesion central to its ganglion (spiral or Corti's) is followed by a central degeneration, at least as far as the next ganglion cell, and a lesion peripheral to the ganglion will result in atrophy as far as the end-organ. The cells of a spinal cord ganglion, however, do not atrophy or disappear after section of either the central or the peripheral nerve trunk. The histologic appearance of the cochlear ganglion in the two cases reported indicates that this ganglion is an exception to the general law.

These two cases clearly indicate that the changes seen in the cochlear ganglion and peripheral nerve fibers are secondary to a lesion of the cochlear nerve central to the ganglion. They give an answer to the argument that a growing tumor in the cerebellopontile angle (in addition to destroying the nerve) may also compress the labyrinthine artery, generally agreed to be an end-artery, and consequently that a poor supply of blood, together with a marked increase in intracranial pressure, could caused the degenerations in the inner ear.

Atrophy of the organ of Corti and the nerve in the osseous spiral lamina (in case 1) was limited to the basal and middle coils of the cochlea. Hearing tests made one month before death showed that the patient could hear some of the low tones but had absolute deafness for all tones above 128 double vibrations. This, Crowe took to be additional evidence that the fibers supplying the apex of the cochlea were intact. If the degenerative changes were due primarily to an interference with the blood supply or to increased intracranial or intralabyrinthine pressure, one would expect to find a more uniform distribution of the lesion in the inner ear as well as an involvement of the vestibular nucleus and end-organs. In case 2, there was a marked increase of intracranial pressure due to an internal hydrocephalus, the effect of which should have been bilateral. The only changes seen, however, were on the right side and were limited to the cochlear branch of the auditory nerve.

It is concluded that the atrophic changes in the spiral ganglion, peripheral nerve fibers and organ of Corti in the two cases were sec-

ondary to a lesion of the auditory nerve central to the cochlear ganglion, and that this ganglion is an exception to the wallerian law of degeneration.

Aural Symptoms in Multiple Sclerosis.—Of 529 patients suffering from multiple sclerosis, 2 patients experienced sudden loss of hearing in one ear.[7] Seven had fairly severe vertigo. The eighth cranial nerve was involved much less frequently than the second. Weakness of facial muscles was observed twice, necessitating differential diagnosis between Bell's palsy and multiple sclerosis.

Tinnitus and Deafness in Intracranial Arteriovenous Fistula.—Horton and Ziegler [8] reported a case of intracranial arteriovenous fistula.

A farmer, twenty-five years previously, suddenly noted complete loss of vision in his right eye, followed a week later by loss of vision in the left eye. He had recovered completely in five weeks. Fifteen years previous to his examination, he began to lose his hearing in the right ear. Later he noticed noises in the right ear, but only on lying down. The noise sounded like the humming of bees, or the singing of a tea-kettle. Ten years previously he had received a blow on the left side of his head; he was not rendered unconscious, and the injury was not considered serious. Three years later he again experienced sudden and complete loss of vision in the right eye, and in a few days marked reduction of vision in the left eye. The right eye never recovered, but there was some improvement in the left. One or two years previous to admission to the hospital, the patient began to lose his hearing in the left ear, in which he noticed noises much like those in the right ear. He could hear the pulse, and whereas heretofore the noises were present only on lying down, they now began to be apparent in all postures.

Examination of the eyes revealed primary optic atrophy. There was bitemporal hemianopia, such as is seen in cases of chiasmal lesions. Roentgenologic examination of the head disclosed erosion in the left temporal region, about 2 cm. in diameter. Neurologic examination revealed horizontal nystagmus of moderate degree, a doubtful left Babinski sign, and bilateral, partial nerve deafness, which was more marked on the right side. A slight thrill could be felt over the external jugular veins, more on the right than on the left. The patient stated that compression of the external jugular veins increased the noises in his head. It was interesting to compare the color of the blood from one external jugular vein of this patient with that from the opposite external jugular vein, as well as with the color of the blood from the vein of the arm of a normal subject. The blood from the patient's jugular veins was bright red; that from the right external jugular vein being slightly brighter red than that from the left. The blood from the vein of the arm of the control had a normal dark red venous color. The color of the mixed venous blood from the patient's jugular veins, when compared with the unmixed venous blood from the normal vein, was sufficient to establish the diagnosis of arteriovenous fistula.

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7. Ziegler, Lloyd H.: Multiple Sclerosis: A Clinical Review and Follow-Up Study, Minnesota Med. **12**:778 (Dec.) 1929.

8. Horton, B. T., and Ziegler, L. H.: Intracranial Arteriovenous Fistula: Diagnosis by Discovery of Arterial Blood in Jugular Veins, Proc. Staff Meet., Mayo Clin. **5**:178 (June 25) 1930.

Acoustic Nerve Atrophy from Roentgen Treatments.—Unterberger [9] reported two cases of permanent injury of the acoustic nerve following roentgen sterilization. In the first case, that of a woman, aged 54, the injury involved both cochlear and vestibular nerves. In the second case, that of a woman, aged 50, it involved only the right cochlear and vestibular nerves. As proof that there was a causal relationship between the artificial suppression of the ovarian function and the disturbance of the acoustic nerve, the author stated: "1. The acoustic nerve symptoms appeared immediately after the appearance of the artificially produced menopause. 2. It developed simultaneously with other characteristic disturbances of the menopause."

Hemorrhages into the Dura and Auditory Nerves, the Result of Otosclerotic Toxemia.—Leicher said that the serum, whole blood, and, to a certain extent, urine, saliva and sweat of otosclerotic patients have a much stronger poisonous effect on plant cells than do similar body fluids from healthy, nonmenstruating subjects. The poisonous effects show themselves in an early death of cut flowers of equal age and in an inhibiting influence on the fermentation of yeast. The poisoning effect is found also in extracts of alcohol, chloroform, ether and acetone, but is absent from a dialysate. The phosphatids are most probably carriers of the poison. The body fluids from otosclerotic patients while menstruating show an increase in the poisonous effect on plants.

Gottlieb's [10] report deals with the effects on guinea-pigs of extracts of the stools from four deaf persons, and from three healthy persons in whom no sign of deafness existed. The feces were obtained in a liquid form after a cathartic containing phenolphthalein. The author concluded that "the stool of the patients whose hearing is becoming progressively worse is undoubtedly toxic to guinea-pigs. It apparently produces hemorrhagic lesions in the cochlea, hemorrhages into nerves, around nerves, and into the dura. These lesions were not found in the controls."

Treatment of Tinnitus by Intraspinal Injections of Dextrose.—Felderman [11] found that the intraspinal introduction of dextrose solution ameliorated tinnitus aurium in three out of five cases. No return of symptoms was noted as late as from four to five weeks subsequently.

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9. Unterberger, S.: Permanent Injury of Acoustic Nerve Caused by Roentgen Sterilization, Ztschr. f. Laryng., Rhin. **19**:132 (Jan.) 1930; abstr., Arch. Otolaryng. **11**:224 (Feb.) 1930.

10. Gottlieb, Mark J.: Lesions of the Cochlea Experimentally Produced in Guinea Pigs by Injecting Fecal Extract from Cases of Progressive Deafness, Laryngoscope **40**:85 (Feb.) 1930.

11. Felderman, Leon: The Newer Treatment of Tinnitus Aurium, M. World **48**:93 (March) 1930.

Three other patients used as controls were not relieved by simple lumbar spinal puncture and drainage. Felderman summarized his observations as follows:

"1. Tinnitus aurium, for which no specific cause exists, is generally caused by increased or fluctuating intralabyrinthine endolymphatic pressure.

"2. The saccus endolymphaticus, a part of the membranous labyrinth, is, in reality, a subarachnoid intracranial structure.

"3. Changes of cerebrospinal osmotic tension should affect endolymphatic pressure."

Central Vestibular Symptoms in Tower Skull.—Formerly of almost exclusive interest to the ophthalmologist, cochlear and vestibular manifestations have attracted the attention of otologists. Barré, Wenger and Alfandary [12] reported a case with disturbances of the second, fifth, eighth (vestibular), ninth, eleventh and twelfth nerves on the right; bilateral pyramidal syndrome of the irritative type (more marked on the left), and a syringomyelic dissociation of sensibility, also on the left.

In 1921, a woman, aged 35, had severe pain in the left arm, followed by icterus and somnolence. In 1923, paralysis of the left arm developed, which was relieved by vaccines. From the winter of 1924 to the spring of 1927, she had severe supraorbital headaches on the right side which extended to the right occiput. Vertigo was present from the summer of 1926, when she experienced heaviness and stiffness in the right thigh. The lips felt as though they were coated with menthol; the tongue seemed heavy during speech. Tinnitus was present in the right ear.

There was bilateral optic atrophy. Touch, pain and heat were felt less on the right side of the face. The seventh and eighth nerves (cochlear) were normal. There was spontaneous nystagmus on looking in all directions, including vertical nystagmus downward. The patient past pointed to the right and downward. She fell to the right. In the caloric test of the right ear there were normal nystagmus and deviation after the use of 25 cc. of water. The caloric test of the left ear required 50 cc. of water and then gave a normal reaction. Turning to the right, there was horizontal nystagmus of small amplitude, lasting thirty seconds; to the left, nystagmus of greater amplitude, lasting sixty seconds. The galvanic test with 10.5 milliamperes showed nystagmus on both sides. Examination of the ninth nerve showed the palate lower on the left and contracting better on this side. The twelfth nerve showed hemiatrophy of the tongue on the right. The reflexes of the acromial and finger flexors were increased on the left. The Mendel-Bechterew sign was positive in the arm. The Babinski sign was positive on both sides. The abdominal reflexes, except the right inferior, were abolished.

Roentgen examination of the head showed multiple deformities: The frontooccipital line of the base formed a right angle, and the occiput seemed soldered to

12. Barré, Wenger and Alfandary: Oxcephaly (Tower Skull): Case Report with Oto-Neuro Ophthalmologic Studies, Rev. d'oto-neuro-opht. **7**:493 (July) 1929; abstr., Arch. Neurol. & Psychiat. **23**:807 (April) 1930.

the atlas; the posterior cerebral fossa was flattened, and the vault was increased in height. This led to an annular construction of the foramen magnum, producing disturbance of the bulbopontile circulation.

Special features are the relatively late appearance of optic atrophy, the multiplicity of the nerves involved, and the existence of vestibular nystagmus.

It is questioned whether or not vestibular disturbances may not be more frequent than they have hitherto been considered. Ruttin studied ten cases of oxycephalia and found most frequently concomitant lesions of the second and eighth nerves. More rarely, there was a lesion of the eighth nerve (cochlear and vestibular) alone. He observed either hypoexcitability or hyperexcitability of the semicircular canals, inexcitability of the vertical canals, or at times inexcitability of all canals. Hyperexcitability was found especially in cases with parietal or occipital hyperostoses, which Ruttin explains as being caused by compression on the cerebellum. Hyperostosis in the occipital region was present in Barré's case, but he and his co-workers think that the pyramidal and sensory disturbances of the fifth, eighth, ninth and twelfth nerves point to a slow sclerotic process due to chronic circulatory disturbances.

Vestibular Symptoms in Degenerative Lesions of the Angle.—In his capacity as medical director of the department of nervous diseases of the Odessa Military Hospital, Rabinowitsch [13] summarized his observations thus: A fragmentary pontobulbar clinical syndrome was often observed in the years 1923-1926, among the young soldiers of the Red Army, men from 21 to 23 years of age. These men chiefly belong to the rural population, their occupation being agriculture; city dwellers, laborers and officials are much more rarely affected.

The principal symptoms are: horizontal rotatory nystagmus, abnormal labyrinth reactions and pyramidal symptoms (including the signs of Bechterew and of Rossolimo). The subjective and objective phenomena of the syndrome are variable and inconstant. (If this syndrome is to be interpreted as multiple sclerosis, it would not predominate among the rural inhabitants, according to German and Swiss writers. Collins [1923] also claimed that multiple sclerosis occurs more frequently in city dwellers and factory workers than in peasants.) No description of similar symptoms is found in the literature.

Vestibular Symptoms in Occlusion of the Posterior Inferior Cerebellar Artery.—The four cases recorded by Thompson [14] portray acute vascular involvement of the lateral area of the medulla oblongata due to occlusion of the posterior inferior cerebellar artery.

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13. Rabinowitsch, S.: Zur Klinik der pontobulbaren Syndrome, Monatschr. f. Psychiat. u. Neurol. **75**:107 (Feb.) 1930.

14. Thompson, Ross H.: Occlusion of the Posterior Inferior Cerebellar Artery: A Clinical Study of Four Cases, Arch. Neurol. & Psychiat. **22**:530 (Sept.) 1929.

The lateral vestibular (Deiters') nucleus lies almost exactly in the lateral angle of the fourth ventricle, close to the restiform body. In a thrombotic process of the posterior inferior cerebellar artery, it is not difficult to see how there may be considerable vestibular irritation, considering the large vascular channels appearing in connection with it. The presence of the syndrome of Avellis, when associated with crossed analgesia and thermo-anesthesia of the trunk and extremities, suggests occlusion of this artery. Symptoms that are referable to the posterior portion of the lateral medulla, especially the restiform body, the vestibular nucleus and the medullary center of the cervical sympathetic, strongly favor occlusion of the posterior inferior cerebellar artery. According to Brunner, occlusion of this artery gives outstanding signs of vestibular involvement.

Vestibular symptoms of varying degree were present in all instances recorded by Thompson.

In one case, spontaneous, transient, conjugate deviation of the eyes to the side of the lesion, together with nystagmus to the opposite side and a vertical diplopia, was observed. The question arose whether the oculogyric manifestations could be attributed to a lesion of the vestibular nucleus and to its arcs alone, or to concomitant higher lesions.

Vestibular and cerebellar symptoms are nystagmus, vertigo and lateropulsion. They were present at least in part in all cases, and may be attributed to involvement within the medulla of the fibers that are concerned with the vestibular pathways connected with the spinal cord, cerebellum and oculomotor nuclei.

TUMORS OF THE CENTRAL AUDITORY AND VESTIBULAR AREAS

Lack of Development of Transverse Sinus the Cause of Absence of Choked Disk in Angle Tumor.—Swift [15] reported the results of an autopsy in a case of tumor of the cerebellar pontile angle, in which during life there were deafness, dizziness and vertigo without any choking of the disk, the absence of the latter being explained by Swift as due to the lack of development of the transverse sinus on the side of the tumor. Thus the growth failed to cause any obstruction to the return venous sinuses as the opposite uncompressed transverse sinus was unduly large.

Swift regarded the transverse and sigmoid sinuses as an "upper circulatory plane" and the cavernous and petrosals as a "lower circulatory plane."

15. Swift, G. W.: The Transverse Sinus and Its Relation to Choked Disk, Arch. Ophth. **3**:47 (Jan.) 1930.

Diminished Hearing Due to a Tumor of the Third Ventricle.—Penfield [16] reported the following case:

A woman had suffered from occipital headaches since an accident to her head at the age of 5. When they were severe, she would have attacks of dizziness. At the age of 28, during an attack, the patient stiffened, fell under the table, was unconscious for several hours and apparently had a convulsion which involved the arms, legs and neck. At the age of 36, she began to show recurring seizures. The attacks would always begin by a request for ice; then her face and arms would flush, tears would roll down the pillow and profuse general diaphoresis would appear. The eyes were open and protruding, the pupils large and the pulse was strong and rapid. Gradually the flushing would fade, the pulse would become weak and slow. The attacks were usually followed by shivering. Hiccup was an annoying and frequent symptom, especially after drinking. During some attacks the patient was unconscious and incontinent. The repeated attacks resembled status epilepticus and could for a time be controlled by sodium bromide. Aside from the attacks, the patient showed a papilledema, diminished hearing bilaterally, and disappearance of headaches after turning the head or changing its position. She had enormously dilated lateral ventricles and absence of air in the third ventricle. The diagnosis of tumor of the third ventricle was made. Necropsy disclosed a tumor in the nature of a cholesteatoma in the anterosuperior portions of the third ventricle. It protruded into the foramen of Monro.

Intracranial Extension of Giant-Cell Tumor Springing from the Cartilaginous Sphenoidal Wing.—Geschickter and Copeland [17] reported the following case:

A white woman, aged 52, suffered from pain near the right ear for three years, with dizziness for six months. Examination showed deafness in the right ear, ataxia with deviation to the right, swelling over the right temporal region, and a negative cerebrospinal fluid. The tumor was partially removed by excision. There was extension about the base of the pons, which was deemed inoperable. Four years after operation the patient had a fully developed hemiplegia due to the continued growth of the tumor.

The microscopic sections of the tumor showed a typical giant-cell growth. The operative notes show that the origin of the lesion was from the posterior body of the sphenoid, extending into the great wing around the carotid groove. This relates the tumor to a center of cartilaginous ossification.

ANATOMY OF SENSATIONS OF TASTE

Lewis and Dandy [18] found no evidence to support the theory that a variable nerve supply conducts the sensations of taste, as first advanced

16. Penfield, W.: Diencephalic Autonomic Epilepsy (Cholesteatoma of Third Ventricle), Arch. Neurol. & Psychiat. **22**:358 (Aug.) 1929; abstr., Arch. Path. **9**:918 (April) 1930.

17. Geschickter, Charles F., and Copeland, Murray M.: Osteitis Fibrosa and Giant-Cell Tumor, Arch. Surg. **19**:242 (Aug.) 1929.

18. Lewis, Dean, and Dandy, Walter E.: The Course of the Nerve Fibers Transmitting Sensation of Taste, Arch. Surg. **21**:249 (Aug.) 1930.

by Krause and more recently sponsored by Harris, who accepted Nageotte's anatomic demonstration that the fifth, seventh and ninth nerves have a common gustatory nucleus in the pons.

Operative observations indicate that but one nerve conducts sensations of taste from the anterior two thirds of the tongue. This is the nervus intermedius (Wrisberg) or the N. glossopalatinus (Hardesty), which represents the sensory portion of the seventh nerve.

The course of the pathway of taste is direct. Stimuli pass from the taste buds through the chorda tympani by way of the geniculate ganglion and the nervus intermedius into the pons.

The following conclusions of Lewis and Dandy were based on the results of intracranial division of the isolated cranial nerves:

"1. Section of the sensory root of the fifth nerve is not followed by any permanent changes in taste. . . .

"2. After intracranial division of the seventh nerve, including the sensory portion (nervus intermedius of Wrisberg, the N. glossopalatinus of Hardesty), taste to the anterior two thirds of the tongue is completely and permanently lost.

"3. Division of the facial nerve, between the geniculate ganglion and the point at which the chorda tympani leaves the facial nerve to pass through the middle ear, is invariably followed by total and permanent loss of taste.

"4. Intracranial division of the glossopharyngeal nerve is followed by complete and permanent loss of taste to the posterior third of the tongue, but there is no effect on taste in the anterior two thirds of the tongue."

GLOSSOPHARYNGEAL NEURALGIA

In an article on the surgical treatment of various types of neuralgia, Craig [19] stated that glossopharyngeal neuralgia is a definite clinical entity, similar to trigeminal neuralgia in its course. The pain probably has its origin in the superior ganglion of the glossopharyngeal nerve. It can be relieved temporarily, and possibly permanently, by extracranial evulsion of the glossopharyngeal nerve and of part of the ganglion, but division of the root intracranially affords permanent cure.

Craig's technic follows:

> Evulsion of the peripheral part of the glossopharyngeal nerve is carried out through an incision in the neck, extending from the tip of the mastoid process inferiorly and anteriorly over the anterior border of the sternocleidomastoid muscle for a distance of 10 cm. Wide dissection of the neck is then done, with partial resection of the sternocleidomastoid muscle, division of the posterior belly of the digastric muscle, and exposure and identification of the spinal accessory, hypo-

19. Craig, W. McK.: Surgical Treatment of the Major Types of Neuralgia, Proc. Staff Meet., Mayo Clin. **4**:84 (March 13) 1929.

glossal, vagus, and glossopharyngeal nerves. Evulsion of the glossopharyngeal nerve is then carried out and the wound is closed. . . .

He adds that the glossopharyngeal nerve is a mixed nerve containing motor and sensory fibers, the latter including those which transmit the impulses of the special sense of taste. The motor element is small and supplies, according to Piersol, only motor fibers to the stylopharyngeus muscle and the secretory fibers to the parotid gland. The sensory fibers are distributed to the mucous membrane of the middle ear, tonsillar fossa, tongue and oral pharynx. Superficially, the glossopharyngeal nerve originates from the rostral end of the posterolateral sulcus of the medulla oblongata, in a line with the tenth and eleventh nerves. Like the vagus, the glossopharyngeal nerve has two ganglions: the dorsal root ganglion, at the superior edge of the jugular foramen, and the trunk or petrous ganglion, in a small depression at the inferior edge of the jugular foramen.

SURGERY IN INTRADURAL SUPPURATIVE LESIONS

Protective Reactions of the Cerebrospinal Fluid System.—In discussing these protective reactions, Diamond [20] described the cells lining the arachnoid, known as endothelial cells (*Häutchenzellen* of Key and Retzius) or, as they are called in this country, mesothelial cells (Weed), as of the low, flat type with large, pale, oval nuclei.

A slow hyperplasia of the arachnoid mesothelium in the form of localized cell clusters or nests is often associated with the presence of corpora aranacea, as first described by Meyer, in 1859, who considered them a product of a "chronic irritation." Trauma and infection were found to be important etiologic factors in the fifty cases studied by Winkelman and Wilson. Weed, Essick and Ayer, however, have shown experimentally that the cells lining the subarachnoid cavity respond to various stimuli (carbon granules, laked blood and infection). These changes consist in an increased size of the cells, their active proliferation, phagocytic activity and the formation of macrophages. Undoubtedly, any other substance harbored within the spinal fluid must act in a like manner on the arachnoid cells (Hassin).

Diamond's conclusions are:

"1. Changes in the arachnoid cells are of rather common occurrence in diseases of the central nervous system.

"2. They are mainly progressive, the most striking being the formation of clusters.

"3. The cell changes vary in intensity and extent according to the type of the disease.

20. Diamond, I. B.: The Spinal Arachnoid: A Note on the Cells in Pathologic Conditions, Arch. Neurol. & Psychiat. **25**:373 (Feb.) 1931.

"4. The changes in the arachnoid may depend on the physicochemical properties of the cerebrospinal fluid."

Obstruction to the Exit of Infected Fluid by Lesions of the Pacchionian Bodies.—The material on which was based the investigation of meningitis, made by Bateman,[21] with special reference to the rôle of the pacchionian bodies, consisted of 1,131 brains from the following subjects: 4 cats, 15 dogs, 6 of which were fetal, 16 white rats, 10 rabbits, 32 sheep; 6 human beings (fetal), 3 infants, 145 normal adults, 152 epileptics and 748 insane persons. Of the human material examined, there were 108 brains that showed in various degrees some exudative process of the meninges.

In inflammation, the cellular exudate is most pronounced in and around the pacchionian granulations. The separate villi become filled with polymorphonuclear leukocytes, with a corresponding increase in the mesothelial cells lining the villus. Some produce obstruction and rupture of the neck of the villus with escape of the fluid and exudate into the subdural cavity.

Bateman cited an example of a chronic abscess of unknown origin, without clinical symptoms, which finally produced an acute unilateral leptomeningitis with occlusion of the pacchionian bodies and almost immediate death. The abscess probably was of months' duration, with frequent subacute exacerbations. The cerebrospinal fluid no doubt was able to carry off the waste products until finally the normal outlet became occluded, when the process scattered.

Bateman also recorded a case of localized meningitis which was diffused by repeated lumbar drainings. The first symptom was a localized cortical irritation. Following drainage of the lumbar sac, the symptoms were relieved. This can be expected in any case of increased intracranial pressure when the fluid pathways are patent. Repeated lumbar punctures gave increasing turbidity of fluid until finally it became so tenacious that it could scarcely run from the needle. The exudate over the left hemisphere was thickest. Repeated withdrawal of the fluid may have caused the original process to extend to the other side.

The following paragraphs are taken from Bateman's article:

"Just a short time ago a neuro-surgeon asked the question, 'Why is it that in so many cases of brain abscess, meningitis, or cerebral contusion one finds upon opening the skull a great amount of sub-dural fluid'? . . . It is a well known fact that in contre-coup injuries there has been considerable movement of the brain itself. The dura is

21. Bateman, J. F.: Meningitis with Special Reference to the Rôle of the Pacchionian Bodies, Ohio State M. J. **25**:970 (Dec.) 1929.

rather firmly attached to the skull and the only way the arachnoid is attached to the dura is by these small tufts of arachnoidal tissue (pacchionian bodies) which project into the longitudinal dural sinuses. The arachnoid is a very fine fibrous tissue, easily ruptured. When these small projections of the arachnoid are ruptured, the cerebro-spinal fluid then escapes into the sub-dural cavity. Here it has no outlet and we get clinical signs of increased intracranial pressure without any demontrable increase in the fluid of the lumbar sac. . . .

"In cases of cortical abscess there is usually associated a meningitis either local or diffuse. . . . Extension of the inflammation is always with the course of the cerebro-spinal fluid and the amount of destruction done depends on how well the exudate and necrotic tissue can be removed. One of the main functions of the cerebro-spinal fluid is to bathe the cells and remove their waste products. If the only means of escape of the fluid becomes occluded our symptoms are markedly exaggerated and death usually ensues before surgical intervention is possible.

"From an anatomical viewpoint any inflammatory process within the meninges may be considered obstructive. Any form of increased intracranial pressure, including serous meningitis, may be considered as obstructive. A swelling or edema in any part of the body likewise may be considered as obstructive. . . . Natural means of escape are provided for all fluids and if there is an increase to the extent of impairment, there must be some form of obstruction within the circulating limits of that fluid.

"A mild meningeal inflammation . . . is very common. Unilateral manifestations of meningeal irritation are usually overlooked, and when they are present one is inclined to think of a brain tumor, abscess or encephalitis.

"Rupture of the pacchionion bodies with escape of cerebro-spinal fluid or exudate into the subdural space is a common finding. This may lead to a purulent or hemorrhagic pachymeningitis interna.

"In a great many instances lumbar drainage causes a localized process to become diffuse. . . ."

Intraspinal Anesthesia in Head Operations.—The action of local anesthetic on nerve tissue depends on an affinity between the anesthetic and the lipoid substances in the nerve, as well as on a physiochemical solubility reaction occurring at the point of application. Lipoidal tissues have an affinity for anesthetics and unite with them with considerable avidity. The anesthetic agent is very rapidly fixed by the lipoid reaction.

Koster and Kasman [22] give the following instructions for anesthesia:

The height of the anesthesia is regulated by the amount of spinal fluid utilized for dissolving the drug. The greater the amount of cerebrospinal fluid with-

22. Koster, H., and Kasman, L. P.: Spinal Anesthesia for the Head, Neck and Thorax: Its Relation to Respiratory Paralysis, Surg., Gynec. & Obst. **49**:617 (Nov.) 1929.

drawn, the greater will be the amount of diffusion and, therefore, the higher the level of anesthesia. This is in accordance with the well known physical law that the diffusion of liquids under pressure is inversely proportional to the pressure. . . .

If anesthesia of the thorax and head is desired, 250 milligrams of neocaine dissolved in 8 cubic centimeters of cerebrospinal fluid injected between the second and third lumbar vertebrae are used. . . . Between the ages of 5 and 8, head anesthesia can be obtained by means of 150 to 200 milligrams of neocaine dissolved in 6 cubic centimeters of fluid. Between the ages of 2 and 5, similar anesthesia may be obtained with 100 to 150 milligrams of neocaine dissolved in 4 cubic centimeters of fluid, and below the age of 2, head anesthesia can be obtained with $\frac{5}{100}$ to $\frac{1}{10}$ of a gram dissolved in 3 cubic centimeters of cerebrospinal fluid.

In the past four and a half years, they have used spinal anesthesia as a routine measure. They have never found its use contraindicated except in cerebellar neoplasms and disease of a suppurative nature of the spinal column or of the overlying tissues at the site, or in the neighborhood, of the injection point.

Koster and Kasman arrived at the conclusion that it is impossible to predict with any degree of accuracy what will happen to the blood pressure after the injection of the anesthetic solution. In some instances there is no appreciable change. Other patients show a maximal fall of about 10 mm. of mercury. A fairly large number have a drop in pressure of between 20 and 30 mm. within ten minutes after the injection, this fall being sustained for approximately three quarters of an hour, the return to normal immediately preceding the return of sensation. In some instances the drop is from 50 to 75 mm. within ten minutes, and within another ten minutes there is a return to about 20 mm. below the normal. In at least 5 per cent of all cases the pressure drops within ten minutes to such a level that no radial pulse can be felt, nor can the pressure be registered by the manometer. It was soon noticed that even without the use of drugs the pressure returns to a higher level within a very short period (approximately ten minutes) if the Trendelenburg position is maintained.

With this form of anesthesia the most frequent complication encountered is postoperative headache, which occurs in about 10 per cent of the cases. In most of these the headache is very mild and is not appreciated until the patient attempts to sit up, when it varies from slight dizziness to slight pain. Occasionally the headache is quite severe, and the patient is very uncomfortable. Magnesium sulphate enema for lowering the cerebrospinal pressure, used in cases of head injury, immediately controls the headache in almost all instances. Six ounces of 50 per cent solution of magnesium sulphate every four hours in an enema is the method employed. One patient who did not respond com-

pletely to this method yielded to a spinal puncture, through which 40 cc. of fluid were removed from the canal. It must be mentioned that the anesthesia has not been perfect in all cases. There have been instances in which the patient has complained of painful sensations.

The experience of the authors with head anesthesia in a fairly large number of cases has convinced them not only that the method is safe but that it is universally applicable.

"Brain Flap" in the Causation of Intracranial Aerocele.—The experiments of Henry and Heathcote,[23] give a complete explanation of the manner in which an aerocele is formed within the cranial cavity. In order for this to occur there must be a compound fracture of the skull, either in the base or in the vault, which tears the dura so as to produce an opening through which air may enter, either from an air sinus or from without.

There must also be a leak of cerebrospinal fluid or a loss of blood, sufficient, separately or together, to reduce the intracranial pressure almost, if not quite, to atmospheric pressure. If, on inspiration, there is now some impediment to the free entry of air to the thorax, this impediment, combined with a low intracranial pressure, will induce the condition of "brain flap."

As the brain recedes from the dura mater on inspiration, air will be drawn in, filling the space that is left, and, being lighter than the cerebrospinal fluid, will tend to rise up through it under the dura.

ARTERIAL ENCEPHALOGRAPHY

Arterial encephalography was devised by Egas Moniz for the purpose of furnishing the localization of cerebral tumors. Pinto [24] advised that arteriography should be carried out on both sides in order to obtain the desired comparison. The value of the procedure has been established in tumors situated in different regions of the brain which depend on the internal carotid for their blood supply. Cerebral arteriography reveals temporal lobe tumors by showing early displacements of the sylvian group vessels often accompanied by obliteration of the "carotid siphon"; however, the arteriograph must be studied in relation to the median line of the skull. For, in small tumors, the posterior temporal artery in descending may resume its normal mode of separation from the sylvian artery, and the posterior parietal artery, which takes its course in a higher position, may divide only in its final portions. The anterior choroid artery, a small branch of the internal carotid, which is usually visible in the inferior portion of the sylvian group, may likewise furnish diagnostic data. If it remains in

23. Henry, Arnold K., and Heathcote, Reginald St. A.: The Causation of Intracranial Aerocele by Brain-Flap, Surg., Gynec. & Obst. **48**:782 (June) 1929.

24. Pinto, A.: Arterial Encephalography, J. de chir. **36**:353 (Sept.) 1930.

its normal position, and if the sylvian group is very high, the tumor is situated in the most central portion of the skull. If the tumor is more superficial or is found on the external portion of the temporal lobe, the artery disappears or rises to a higher position, in accompanying the sylvian group. Frontal tumors are revealed by the curvature of the carotid siphon and by the lowering of the first portion of the sylvian group. In one of the cases studied, this group of arteries was lowered so as to extend nearly to the roentgenographic area representing the petrous bone.

Occipital tumors manifest themselves through displacement of the terminal branches of the arteries of the sylvian group or by the modification of the circulation in the occipital portion of the brain.

Moniz [25] added to his earlier reports on his method of arteriography, by injection of iodine solutions into the internal carotid. He no longer exposes the carotid operatively, but injects directly through the skin. To the iodine solution he now adds strontium bromide, a 7 per cent solution. At the present time an intracarotid injection of from 4 to 6 cc. of 25 per cent sodium iodide, with 7 per cent strontium bromide, is used. In children after the fifth year of life, 2 cc. are often sufficient.

SUPPURATIVE INTRACRANIAL COMPLICATIONS IN GENERAL

Several of Stewart and Fraser's patients with extradural abscess [26] in the middle fossa had symptoms suggestive of a lesion of the posterior fossa, e.g., spontaneous nystagmus, giddiness, falling, dysdiadokokinesia or slurring speech; while only in four cases, the extradural abscess in the middle fossa was associated with a lesion of the labyrinth or serous meningitis sufficient to explain the presence of these symptoms.

Temporal Lobe Abscess: There were nine cases of this condition. Headache was a marked feature in seven and vomiting in six. The temperature on admission was subnormal in four; fever was present in five patients.

As regard localizing symptoms, tenderness on percussion of the temporal region was noted in two, dilatation of the pupil on the affected side in two, and sensory aphasia in three of the four patients with left-sided abscess. In one case contractions of the facial muscles on the homolateral side were noted, and in another, "clawing" of the homolateral side of the head.

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25. Moniz, Egas: Encephalographia arterialis, Rev. otol. y cir. neurol. **4**:276, 1929.

26. Stewart, J. P., and Fraser, J. S.: The Mastoid, Labyrinthine and Intracranial Complications of Chronic Suppurative Otitis Media (Part III consists of reports on forty-seven cases of intracranial complications), J. Laryng. & Otol. **45**:243 (April); 305 (May) 1930.

In three cases signs of meningitis developed only after an operation for brain abscess. In two of these cases there was a flow of cerebrospinal fluid from the abscess, pointing to rupture into, or leakage from, the lateral ventricle.

Of the nine patients, three recovered and six died.

Cerebellar Abscess: There were six cases in this group. The labyrinth was apparently healthy in three and functionless in two. In all, subnormal temperature and slow pulse were present before operation. Two of the patients had slow and slurring speech. Giddiness and falling to the diseased side were prominent in two cases. Spontaneous nystagmus to the diseased side was present in two, and to the healthy side in one, while in the other three the direction of the nystagmus varied. Tenderness on tapping over the cerebellum was found in only one case. The presence of dysdiadokokinesia was of great value in four of the six cases, while the finger-to-nose test was poorly performed on the diseased side in two cases.

In one case the patient called all objects shown to him "pens," a fact which, unfortunately, led to exploration of the temporal lobe before the cerebellar abscess was opened.

In one patient spontaneous respirations ceased during the operation but recommenced after the abscess had been opened.

Of the six patients, two recovered and four died. One of those who recovered had a ravenous appetite during convalescence. (I have found that in all cases in which the appetite is good, prognosis is favorable.) In three of the four fatal cases, more than one abscess was present in the cerebellum.

Purulent Leptomeningitis: There were six cases in this group. Purulent labyrinthitis was present in one and latent labyrinthitis in three. In two, the inner ear was healthy.

Four patients had an anxious expression; Kernig's sign was positive in all. Spontaneous nystagmus to the healthy side was present in one, to the diseased side in another, and in various directions in three.

In two patients who recovered there were abundant pus cells in the cerebrospinal fluid, but no growth was obtained on culture.

Cheng [27] reported eight cases (from the division of neurology, Peiping Union Medical College) in which the following conditions were found: (1) suppurative nonhemolytic streptococcus cerebrospinal meningitis; (2) acute mastoiditis and hemolytic streptococcus meningitis; (3) multiple abscesses, arachnoid abscess, abscess of the cerebellum, suppurative Staphylococcus aureus cerebrospinal meningitis

27. Cheng, Y. L.: Intracranial Complication of Suppurative Middle Ear Disease: Eight Cases, China M. J. **43**:1064 (Nov.) 1929.

and brain abscess; (6) brain abscess; (7) cerebrospinal meningitis and thrombosis of the cavernous sinus; (8) thrombosis of the lateral sinus and hemolytic streptococcus bacteremia.

Summarizing the findings in these cases, Cheng said that in 7.4 per cent of the cases of otitis media intracranial complications develop, usually following an acute exacerbation of a chronic process. Pulse rate is slow in brain abscess. Elevation of blood pressure, slowing of respiratory rate and choked disk are not usually found.

Patients with brain abscess are very irritable in the early stage of the disease but later become drowsy. In purulent meningitis there may not be a very high cell content in the spinal fluid, but in brain abscess the count may be very high in the early meningitic stage.

Cairns [28] commented on the poorer results in treatment of otogenic brain abscess during recent years in comparison with the much better results obtained by Macewen over thirty years ago. Macewen had success in 80 per cent of his cases; while, according to Neumann, the best at present is 62 per cent. Cairns considered that one reason may be due to the fact that Macewen never used a mallet but always opened the mastoid process with hand burs. Cairns expressed the opinion that perhaps the hammering by its intermittent percussion of the skull may produce a disturbance that tends to promote the spread of the infective process.

He also stated that arachnoiditis (serous meningitis) simulates cerebellar rather than temporosphenoidal abscess. It follows either acute or chronic mastoditis, especially when the labyrinth has been infected. The main clinical feature is papilledema, often of extreme degree. The headache may be so slight that the condition is not recognized until sight is seriously impaired.

Cairns believed that a cerebellar decompression, performed before destruction of the optic disks is far advanced, will preserve vision. It should be carried out in every case in which the papilledema is at all severe or in which there is any sign of lowered visual acuity. In one patient who was practically blind, the return of vision was dramatic after this operation. Cairns gave the history of a boy in whom meningo-encephalitis simulated brain abscess.

Aboulker and Badaroux [29] are of the opinion that during exposure of the mastoid process, puncture of the brain should be done only when there are changes such as dural meningeal fistulas.

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28. Cairns, Hugh: Abscess of the Brain, J. Laryng. & Otol. **45**:385 (June) 1930.

29. Aboulker, H., and Badaroux, A.: Cranial Trephining at a Distance: General Method of Diagnosis and Treatment of All the Intradural Complications Derived from the Ear, Arch. internat. de laryng. **9**:385 (April) 1930.

They note that subdural abscess is common but is often unrecognized, as it can rarely be differentiated symptomatologically from brain abscess and diffuse meningitis. In the presence of subdural abscess it is necessary to make wide incisions of the dura.

Incident of Intracranial Complications in Relation to Mastoidectomies.—A collection of the material from the Oto-Laryngological Clinic of the University of Bordeaux [30] for the years 1925-1928 shows that among 283 mastoidectomies and radical operations there were 14 cases of meningitis, 9 cases of brain abscess, 17 cases of sinus phlebitis, and 3 cases of septicemia. Two cases of frontal lobe abscess were not recognized. In two cases spontaneous nystagmus toward the affected side, and vertigo associated with labyrinthine hypo-irritability were suggestive of cerebellar abscess, although the suppuration was in the middle fossa. In operating for brain abscess Portmann prefers chloroform anesthesia.

Intracranial Complications in Relation to Chronicity of the Primary Focus.—Warren [31] said that about one of every four cases of chronic infection and one of every nine cases of acute infection of the middle ear present an intracranial complication. The mortality is practically the same, though slightly higher in acute cases. In dealing with a case in which an intracranial lesion arising from an infected ear is suspected, Warren thinks that the following classification should be kept in mind: (*a*) circumscribed, (*b*) diffuse and (*c*) a combination of these two. Abscess of the brain would come under *a*, meningitis under *b*, and sinus thrombosis under *c*. The only important landmark on the anterior surface of the petrous bone is the eminentia arcuata, below which lies the semicircular canal. The convexity of the superior semicircular canal is in proximity to the floor of the middle fossa. Infection passing through this route produces an abscess of the temporal lobe. The important landmarks on the posterior surface are: (1) the aquaeductus vestibuli, the communication between the endolymphatic space of the inner ear and the endodural space; (2) the aquaeductus cochleae, the communication between the perilymphatic space of the inner ear and the subdural space of the brain; (3) the fossa arcuata, which is rudimentary in human beings and contains only connective tissue, and (4) the internal auditory meatus.

Suppuration which goes through the inner ear into the brain through pathologic fistulas produces abscess, and that which passes from the

30. Faucher, H.: Considerations on Brain Abscess of Otogenic and Rhinogenic Origin, Rev. de laryng. **50**:403; 443, 1929.

31. Warren, W. C., Jr.: Intracranial Complications in Purulent Otitis Media, Arch. Surg. **18**:1552 (April) 1929.

inner ear into the brain through natural ways produces diffuse meningitis with the exception of infection that goes through the aquaeductus vestibuli, which usually produces abscess.

Streptococcus is the most common organism, a pure culture being more frequent than a mixed one. The classification, limitation and structural arrangement of brain abscesses depend on the texture of the brain substance and the mode of entrance of the infection. The direct spread of the infection by way of the tegmen tympani causes the greatest number of temporosphenoidal abscesses, while the majority of cerebellar abscesses are thought to be embolic in origin, the infection being carried through the blood stream.

The majority of brain abscesses are situated near the diseased area of bone that forms the immediate pathway of intracranial infection, but in a minority of cases there is a considerable distance between the osseous lesion and the resulting abscess. Infection of the middle ear may heal entirely before symptoms of an abscess develop. The abscess, having become encapsulated, may remain without producing symptoms for many months.

DIAGNOSIS OF INTRACRANIAL COMPLICATIONS IN GENERAL

Cerebrospinal Fluid in Diagnosis.—According to Steiner,[32] the *inorganic phosphorus content of the cerebrospinal fluid* in healthy persons varies between 1.4 and 1.6 mg. per hundred cubic centimeters. In meningitis, the inorganic phosphorus content is increased, varying from 1.7 to 3 mg. per hundred cubic centimeters. This fact is of value in differential diagnosis, as in brain tumor and acute encephalitis the inorganic phosphorus content is decreased or normal.

Clinical Value of Variations in the Cell Content Obtained at Different Levels.[33]—In several patients with various degrees of otogenic meningitis, lumbar puncture was performed in such a way that the spinal fluid was received in a series of test tubes. The cells were then counted in each of these portions. The result was graphically registered in a rectangular coordinate system in which the abscissa represented the quantity of the spinal fluid in the individual portions, while the cell contents were noted as vertical lines over the middle of the abscissa parallel with the ordinate axis. The results may be summarized as follows: 1. In otogenic meningeal irritative states, the pleocytosis

32. Steiner, B.: Inorganic Phosphorus Content of Cerebrospinal Fluid in Meningitis, Brain Tumor and Acute Encephalitis, Jahrb. f. Kinderh. **124**:188 (June) 1929; abstr., J. A. M. A. **93**:1031 (Sept. 28) 1929.

33. Westergaard, A.: Variations of Pleocytosis in the Different Portions of the Spinal Fluid in Otogenic Meningeal Irritative Conditions, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **120**:164, 1929; Zentralbl. f. Hals-, Nasen- u. Ohrenh. **15**:55, 1930.

often fluctuates considerably in the different portions of the spinal fluid. 2. These fluctuations are the expression of a distribution of the cells in the spinal fluid which can be clinically utilized. 3. An ascending curve is found when the meningeal irritation increases in the cranial cavity. The more rapid this increase, the more abrupt is the curve. 4. A descending curve is found when the process decreases. 5. A curve taking a horizontal course is found when the process proceeds so slowly that the fluctuations of pleocytosis are compensated by the current in the spinal fluid. 6. It is futile to speak of a definite number of cells in the spinal fluid. 7. In order to estimate the pleocytosis, it is accordingly not sufficient to examine a single accidentally picked portion of the spinal fluid. At any rate, the first and the last portion must be included in the examination.

Differential Diagnosis Between Lesions of the Labyrinth and of the Cerebellum by Vestibular Symptoms.—Differential diagnosis of labyrinthine and cerebellar lesions can be aided by a comparison of the deviation of the eyes and the ocular ataxia, although they may be obscured by lesions affecting both (Wilson [34]). Lesions of the labyrinth, which result in loss of function, are shown by a slow deviation of the eyes to the side of the lesion with a quick return to the opposite side. Lesions of a lateral lobe of the cerebellum result in slow deviation to the side opposite the lesion and quick return to the side of the lesion. In cerebellar lesions, there is an ataxia of the eye muscles on fixation, which is most marked on looking to the side of the lesion.

Further observations made by Wilson are: Loss of labyrinthine function is quickly compensated, while loss of cerebellar function is slowly, if ever, fully compensated. Intracranial lesions near the midbrain are marked by vertical nystagmus and dissociation of eye movements, such as unilateral nystagmus, abnormal caloric reactions, et cetera. Intracranial lesions above the midbrain do not interfere with the vestibulo-ocular reflex but at times give qualitative and quantitative changes in the vestibulo-ocular reflex from caloric reaction and rotation. Cerebral inhibition can control nystagmus (certainly the quick phase). This suggests a center above the midbrain receiving information from the periphery.

Nystagmus in the Diagnosis of Intracranial Lesions.—Grahe's [35] contribution is based on twenty-one cases. The first group comprises thirteen gunshot injuries, and the second group, two tumors verified by autopsy findings; the third group is represented by a nonotogenic

34. Wilson, J. Gordon: Contributions of Otology to Neurology, Arch. Otolaryng. **11**:265 (March) 1930.

35. Grahe, K.: Otoneurological Experiences in Affections of the Middle Cranial Fossa: Temporosphenoidal Lobe Lesions, Monatschr. f. Ohrenh. **64**:3, 1930.

temporal lobe abscess; the fourth group comprises three diseases of the temporal lobe of obscure etiology. Grahe pointed out that a characteristic nystagmus does not exist in temporal and parietal lobe diseases. In my opinion, a loss of the rapid component of the nystagmus often occurs in temporal lobe diseases. Grahe observed no loss of the rapid component, but repeatedly noted a difference in the frequency of the ocular twitching on comparison of the two sides. Eight patients showed a less active nystagmus toward the affected side (38 per cent of the total figure); two patients, vice versa, showed slower beats toward the healthy side (9.5 per cent). In a certain sense, Grahe is thus enabled to confirm my statements.

Experimentally Produced Vestibular Symptoms.—In experimenting with alcoholism, Dukhovnikova and Robinson [36] came to the conclusion that chronic alcoholic intoxication affects systems controlling specific functions. It picks out the nerve elements dealing with the functions of position and equilibrium, the vegetative centers, and finally the second and fifth layers of the cortex. The pathologic changes in the central nervous system are essentially of a regressive character with little glial reaction. As these changes are found in all other types of intoxication there is nothing specific about them. The organs most affected by alcohol were the cerebellum, Deiters' nucleus, the red nucleus and the corpora quadrigemina. There were also small areas of degeneration in the vicinity of the lateral ventricles.

Diagnosis of Suppurative Lesions by Examination of the Visual Tract.—Some time ago, I [37] came to appreciate the importance of repeated examination of the visual fields in cases in which intracranial complications were suspected. Only the cases of patients whose mental condition allowed cooperation are reported. Of thirteen patients with brain abscess, seven recovered. Two cases of bulbar cisternal meningitis, four cases of edema of the brain, and numerous instances of localized meningitis of the middle fossa furnish the material. It is my purpose to show that (1) edema of the brain may be diagnosed early by the presence of hemianopic indentations of the fields; (2) papilledema at times is of some localizing value in suppurative diseases of the brain; (3) localized meningitis of the bulbar cisterna prevents a symptom complex, consisting of both posterior and middle fossa symptoms with bitemporal hemianopic indentations, associated with a

36. Dukhovnikova, L. M., and Robinson, J. A.: Pathologic Changes in the Central Nervous System in Experimental Alcoholism, Collected Papers, Moscow State Neuro-Psychiatric Dispensary, 1928, vol. 1, p. 56; abstr., Arch. Neurol. & Psychiat. **23**:169 (Jan.) 1930.

37. Eagleton, Wells P.: Localizing Value of Ophthalmic Examination in Suppurative Diseases of the Brain, J. A. M. A. **92**:713 (March 2) 1929.

peculiar semistupor and paralysis of the sixth nerve, and (4) oculomotor or group dysfunctions may be studied through their connections with the vestibular apparatus.

Swelling of the nerve head from suppuration around the optic foramen from spheno-ethmoid disease causes neuritis, central scotoma with rapidly failing vision, and a sign that has not been described previously, viz., *pain on pressing the eyeball back into the orbit,* as well as on movement of the eyes.

In tumor, the brain is subjected to gradually increasing pressure; in vascular disease, localized areas of cerebral tissue suffer from nutritional disturbances, and in septic disease the infected area is subjected to an invading edema. Brain abscess and septic infarcts offer acute insults to cerebral tissue. Both are largely edematous processes. I believe that cerebral edema involving the homolateral optic tract causes the visual defect. The changes in the visual fields in edema of the brain (caused by infection, abscess, septic infarcts and retrothrombophlebitis) have the following characteristics: 1. They are rapidly changing. 2. The hemianopic indentation often precedes by days or even weeks outstanding neurologic symptoms. It may be sufficient to localize the lesion. 3. The field defect is not recognized by the patient.

Encephalography in Brain Abscess.—A Polish author, Laskiewicz,[38] described two illuminative cases: The first is that of a temporal lobe abscess in a man of 25 years; the abscess had ruptured almost without symptoms into the lateral ventricle, causing a distinct pneumo-encephalon. The ventricular fistula was demonstrated by the utilization of iodized poppy seed oil, 40 per cent, in roentgenography. The cause of death was progressive encephalomalacia with diffuse meningitis. In the second case, that of a youth, aged 19, the author described the roentgenologic picture as revealing a progressive temporal lobe abscess, with rupture into the lateral ventricle, due to chronic middle ear inflammation.

Frazier and Glaser [39] summarized their study of the use of iodized rape-seed oil for cerebrospinal photography by the following statement: "As the result of experiments with various substances for cerebrospinal photography, the iodized oils seem at this time to offer the greatest promise. Iodized rape-seed oil (campiodol) diluted with ethyl olive oil has been found to be the most satisfactory of the oils investigated.

38. Laskiewicz, A.: Roentgenological Picture of Otogenic Intracranial Complications, Polska przegl. otol. **7**:39, 1930.

39. Frazier, C. H., and Glaser, M. A.: Iodized Rape-Seed Oil (Campiodol) for Cerebrospinal Visualization, J. A. M. A. **91**:1613 (Nov. 24) 1928.

Physically and biologically it seems to be ideal, and its practical value after spinal, cisternal, vascular and ventricular injection has been demonstrated."

Suppurative Sinusitis Simulating Cavernous Sinus Thrombophlebitis.—The picture presented in cases such as the three reported by Potts [40] closely simulates cavernous sinus thrombosis. The eyelids are swollen from slight redness to complete closure of the lids. Exophthalmos may be present; the excursion of the eyes is limited or fixed. In pseudocavernous sinus thrombosis the orbital swelling is due to cellulitis as a direct extension from some nasal sinus, and frequently involves only one eye; if bilateral, the swelling of the two eyes usually occurs at nearly the same time. In cavernous sinus thrombosis the swelling begins in one eye, and as the infection extends, the other eye is affected. In the pseudotype the spinal fluid is normal unless there is intracranial extension.

Internal Hydrocephalus from Middle Ear Suppuration.—In the course of a slight middle ear suppuration,[41] there suddenly appeared general symptoms of brain pressure, choked disk, bilateral deterioration of vision and paralysis of the third, fourth and sixth nerves. Diabetes insipidus was also present, which pointed to involvement of the region of the sella turcica. The diagnosis was internal hydrocephalus, the third ventricle becoming distended downward. Lumbar punctures were without effect, but ventricular punctures resulted in rapid improvement.

Typhoid Fever Simulating Suppurative Disease.—Wagner [42] reported two cases in which otogenic intracranial complications were suspected. In the first case the patient was suffering from epitympanal suppuration and chills, and in the second, from sepsis and remittent fever. Bilateral otorrhea and profuse offensive pus with mesotympanal perforation occurred in the latter case. Operation revealed no otogenic complication. Autopsy, three days later, showed the presence of typhoid fever with pulmonary edema and degeneration of the parenchymatous organs.

Cure of Arachnoiditis Leads to Obesity.[43]—A boy, aged 12 years, was admitted to the hospital with acute otitis media; the temperature subsided after paracentesis. After three days, the pulse count dropped to 48; there were tenderness over the

40. Potts, J. B.: Brain Abscess and Meningitis Secondary to Acute Paranasal Sinusitis, Ann. Otol., Rhin. & Laryng. **38**:329 (June) 1929.

41. Katzschmann: Acute Internal Hydrocephalus After Middle Ear Suppuration, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **17**:463 (Dec.) 1929.

42. Wagner, K.: Simulated Otogenic Intracranial Complication, Orvosi hetil. **74**:648 (July 5) 1930.

43. Brain, W. Russell: Arachnoiditis Following Acute Mastoiditis and Leading to Obesity, Proc. Roy. Soc. Med. (Sect. Neurol.) **22**:527 (Jan. 9) 1930.

right mastoid process, bilateral papillary edema and nystagmus. These symptoms persisted after a perisinus abscess was opened. A fortnight later, paresis of the left externus rectus developed. In another ten days, there was right-sided facial paresis. Lumbar puncture showed a pressure of 460 mm. and three degenerated lymphocytes per cubic millimeter. No Kernig's symptom was noted. During convalescence, the boy gained 19 pounds (8.6 Kg.) in weight in the course of two months.

Abducens Paralysis Cured Without Opening the Mastoid.—A Japanese author, Watanabe,[44] described a case of otogenic abducens paralysis, in which cure was obtained without the mastoid process being opened.

The patient was a woman of 25 years with a negative Wassermann reaction. Acute left-sided otitis media was followed some time later by left trigeminal neuralgia, which persisted for fifty days. Left-sided abducens paralysis appeared twenty-five days after the beginning of the trouble and disappeared in another twenty-five days. Soon after the left-sided middle ear inflammation had healed, acute middle ear suppuration with Gradenigo's symptom-complex developed on the right side, but likewise disappeared in a very short time.

BRAIN ABSCESS

Surgical Pathology of Brain Abscess.—My article[45] is based on a description of fourteen selected brains removed from patients who had died from suppurative intracranial complications. "Each brain teaches a distinct surgical lession." A discussion of the developmental relationship between the intermedullary blood supply and the meninges, as well as the points of vulnerability by venous channels and perivascular spaces —as the chief routes of infection in brain abscess—is followed by an account of gross hemorrhages in brain abscess, as shown in a number of illustrations. Specific surgical lesions in intradural suppuration are infarcts and subdural space abscesses. The location of the primary focus of infection in certain areas causes an extension into the brain by definite paths. Consequently, great importance is attached to the exact site of the original infection in the middle ear. My operative results have been good in cerebellar abscess and in abscess of the frontal region, but disappointing in temporosphenoidal lobe abscess. The results in meningitis have been very encouraging: Of the last 107 patients operated on, 62 (about 60 per cent) have recovered.

Spontaneous Absorption of Infarct in the Brain Simulating a Brain Abscess.[46]—In the case of a child of 9 years, the performance of

44. Watanabe, T.: A Case of Bilateral Otogenic Abducens Paralysis, Otologia (Fukuoka), Japan **3**:10, 1930; abstr., Zentralbl. f. Hals-, Nasen- u. Ohrenh. **15**:151, 1930.

45. Eagleton, Wells P.: Brain Abscess from the Standpoint of the Otolaryngologist, Tr. Am. Acad. Ophth. **34**:64, 1929; Laryngoscope **40**:336 (May) 1930.

46. Hlaváček, V.: Cured Otitis, with Later Cerebral Symptoms, Časop. lék. česk. **2**:1041 (July 11) 1930.

antrotomy on the right side on account of subacute otitis with mastoiditis was followed one week later by symptoms of intracranial complication, in the form of epileptiform attacks, with clonic-tonic spasms of the upper and lower contralateral extremities and the left half of the face, paresis of the left abducens, and loss of consciousness. Such symptoms can be explained by the presence of perivenous infarcts: When the infarct becomes infected, an abscess will form or meningitis will supervene. Absorption occurs in many instances, as in the case reported.

Importance of a Chronological History in the Diagnosis of Brain Abscess.—The history is perhaps the one most important factor in any diagnosis.[47] It is invaluable in brain abscess, in the early stages of which so much depends on subjective findings.

The only data obtained in the case reported by Hill referred to severe headache for three weeks, and paralysis of the left arm and leg for one week.

Necropsy disclosed a thrombosed left lateral sinus and a localized area of meningitis below the right frontal region. On sectioning, a fairly large nonencapsulated abscess was found in the posterior part of the frontal lobe. Death evidently occurred from encephalitis. The lesion was apparently an acute metastatic abscess of the frontal lobe resulting from a contralateral otogenic sinus thrombosis.

In an attempt to obtain further information as to history, Hill is trying the experiment of sending out a questionnaire with a letter to the physician who refers any patient with an ear condition. This calls attention to the question of intracranial complication and requests the physician to check any of the enumerated symptoms or signs he may have noted in the case, giving their time of onset.

The diagnosis of brain abscess is rarely made in the first stage of its development, but reasonable grounds for suspicion after encapsulation may be found in the history of a previous infection of the mastoid or paranasal sinuses, provided, of course, that there is evidence of a localized intracranial disturbance. The history of a chill, emphasized by McEwen, has been so frequently lacking in Coleman's [48] cases that he does not consider it an important feature in the diagnosis. Symptoms of the first stage are those of diffuse encephalitis. Coleman reported twenty-eight cases, with but nine deaths; two of the patients who recovered had hemiplegia.

Symptoms of Brain Abscess.—Short histories of seventeen consecutive cases of temporal lobe abscess of otitic origin in which opera-

47. Hill, F. T.: The Importance of the History in the Diagnosis of Brain Abscess, Ann. Otol., Rhin. & Laryng. **39**:595 (June) 1930.

48. Coleman, C. C.: Brain Abscess, J. A. M. A. **95**:568 (Aug. 23) 1930.

tion was performed are recorded by Fraser and Bloomfield.[49] The labyrinth functioned in thirteen cases and was inactive in two. Headache was a marked feature in fifteen cases, and vomiting in thirteen. Seven patients showed subnormal temperatures, eight had fever, and in two the temperature was normal.

The pulse was slow in five cases and rapid in eight; ten of the patients were drowsy or even comatose on admission. In four cases, slight delirium alternated with drowsiness.

There was tenderness on percussion of the temporal region in four cases, dilatation of the pupil on the affected side in three and sensory aphasia in only four of the eight patients with left-sided abscess. One patient with a right-sided abscess suffered from sudden loss of speech. Of eight patients examined, double optic neuritis was noted in two, and congestion and swelling of the disk on the affected side in one, and one had slight edema of both disks, most marked on the diseased side.

Meningitis was present in six patients on admission; only one recovered. In six others, meningitis developed after the first operation; all of these patients died.

Headache in Suppurative Lesions.—Headache[50] in extradural abscess is generally mild, and never so severe as in brain abscess. A localized serous meningitis causes severe headaches, resembling the pain of brain abscess. In diffuse meningitis, the headache is extremely severe and increased by light and noise. It is sometimes restricted to the affected side, but is generally diffuse. The headache is the earliest and most constant symptom of brain abscess. It is rarely absent. It is at first moderate; the patient speaks of a band around the head, with a sensation of weight. Suddenly the pain becomes more severe and is increased by movements and efforts. Certain positions of the head may diminish or increase the pain. The site of the headache corresponds fairly accurately to the seat of the abscess, the headache in temporal lobe abscess being localized over the vertex; in cerebellar abscess it is localized in the occiput. Rapid emaciation and muscular wasting is pathognomonic of brain abscess.

Brain Abscess with Subsidence of Aphasia After Negative Punctures.—Hinsberg[51] reported two cases in which symptoms subsided after negative brain punctures.

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49. Fraser, J. S., and Bloomfield, B. B.: Temporal Lobe Abscess, Eye, Ear, Nose & Throat Monthly **9**:284 (Aug.) 1930.

50. Portmann, G., and Retrouvey, H.: Headache in Otogenic Cerebral and Cerebellar Abscess, Rev. de laryng. **50**:537, 1929.

51. Hinsberg, V.: Rare Observations in Brain Abscess, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **17**:421 (Jan.) 1929.

Case 1.—The patient was suffering from typhus exanthematus in the presence of left-sided chronic otitis media. He presented the typical picture of a left-sided temporal abscess, with right-sided hemiplegia, aphasia and confusion. Radical operation disclosed a cholesteatoma. The tegmen was unchanged. The dura of the middle cranial fossa was widely exposed; brain punctures gave negative results. At first the mental confusion and the aphasia disappeared, and the hemiplegia improved so that the patient could walk. After about four weeks of complete well-being, a large encapsulated temporal lobe abscess was accidentally discovered. At the end of a year the right-sided paresis still continued.

Case 2.—The patient had chronic left-sided otitis media with vertigo and vomiting. Radical operation exposed a large cholesteatoma. On the following day, the lumbar fluid contained 132 cells. A dead labyrinth was opened. The irritative labyrinthine and the meningeal symptoms subsided. Two days later, sensory aphasia developed. Brain puncture gave a negative result. Incision of the dura caused a further recession of the meningitic and labyrinthine irritative symptoms. Repeatedly unsuccessful punctures at last located a large temporal lobe abscess situated very far forward. The patient recovered.

Hinsberg stated that it is difficult to explain the retrogression of the paralysis or the aphasia; apparently the exposure of the dura and the unsuccessful brain puncture in one case, and the incision of the dura in the other, had a decompressing effect.

Hinsberg also reported the case of a man, aged 67, who had cerebellar abscess, in the presence of a middle ear carcinoma, and a temporal lobe abscess following a gunshot wound in the left mastoid process thirteen years previously. Autopsy revealed a large abscess in the left temporal lobe and in the left half of the brain stem. The appearance of a brain abscess such a long time after the ear injury is noteworthy, especially as no diseased bone was found at the time of the operation.

Brain Abscess (Temporosphenoidal) with a Positive Wassermann Reaction.—In a case of idiopathic abscess in the right temporosphenoidal region, reported by Lemierre and Thurel,[52] the Bordet-Wassermann and colloidal gold tests were positive. Hypertension was present. The cerebrospinal fluid obtained by puncture was turbid and contained many altered leukocytes. Bacteriologic examination gave negative results, and the cerebrospinal fluid rapidly regained a normal aspect.

The patient suffered from intense headache in the fronto-orbital region, together with photophobia and vomiting. There was a constant desire for sleep, with a retardation of mental processes. There were a slight stiffness of the neck and paralysis of the right oculomotor nerve. Necropsy revealed slight exudation on the meninges at the base, with an encysted abscess, the size of an egg, in the right temporosphenoid region. The abscess was encapsulated by a membrane which bulged on one side. Cultures from the pus revealed staphylococci. The other organs of the body did not show pathologic changes.

52. Lemierre, A., and Thurel, R.: Idiopathic Abscess in Right Temporo-Sphenoid Region of Brain, Bull. et mém. Soc. méd. d. hôp. de Paris **53**:974 (July 15) 1929; abstr., J. A. M. A. **93**:1598 (Nov. 16) 1929.

UNUSUAL TYPES OF BRAIN ABSCESS

Brain Abscess from the Petrous Apex.—Leidler [53] reported a case (no. 3) of an abscess which he thinks probably was caused by suppuration in the apex of the petrous pyramid, as it was associated with a bilateral organic abducens paralysis.

Brain Abscess from Tonsillar Infection.—The occurrence of cerebral abscess as a direct sequel of tonsillar infection is rare. Such a case is described by Housden,[54] who stated that there is little or no reference to such cases in the literature.

Johnson [55] reported the following case:

A boy of 7 became suddenly ill, from severe acute tonsillitis. Five and one-half weeks after this attack, the tonsils and adenoids were removed. After a normal convalescence of five days, there was a sudden sharp rise in temperature accompanied by a rigor. Five days later a slight left-sided weakness developed into a complete left-sided hemiplegia. The lumbar fluid was sterile.

Postmortem examination showed an abscess of the right hemisphere, 2½ inches in diameter, situated posteriorly to the fissure of Rolando, ⅛ of an inch below the surface and extending downward from the vertex.

The tonsillar fossae were found to be healing.

There are two theories of the production of cerebral abscess after tonsillectomy. One is that infected pulmonary emboli result from the sepsis in the tonsillar bed; the other, that septic material is inhaled while the patient is under the anesthetic. The cerebral abscess is secondary to one of these conditions.

Cadbury and Siddall [56] also reported a case of multiple unilateral cerebral abscesses following shortly after tonsillectomy.

Tonsillectomy was performed, procaine hydrochloride being used as an anesthetic. Convalescence proceeded normally for five days, when sudden epileptiform convulsions occurred. At autopsy, abscesses were found in the right frontal lobe, one abscess in the right parietal lobe, and two in the right temporal lobe.

The position of the abscesses would suggest that they were caused by direct transmission of septic material through the internal carotid artery from the right tonsil.

Metastatic Brain Abscess from the Ear.—Collins [57] classified brain abscesses under three headings, viz., adjacent, traumatic and metastatic, and reported two cases belonging to the third group. While the second

53. Leidler, R.: Three Rare Cases of Otogenic Gravity Abscesses, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:187 (Aug.) 1929.

54. Housden, E. G.: Brit. M. J. **1**:1158 (June 29) 1929.

55. Johnson, W. C.: Cerebral Abscess Following Tonsillar Infection, Brit. M. J. **2**:13 (July 5) 1930.

56. Cadbury, W. W., and Siddall, A. C.: Cerebral Abscess Following Tonsillectomy, China M. J. **44**:910 (Sept.) 1930.

57. Collins, E. G.: Brain Abscess with Unusual Features, Brit. M. J. **1**:438 (March 8) 1930.

of these cases is of aural origin, pathologically and clinically it falls into the metastatic class. The largest collection of pus was contralateral to the original focus. The case was diagnosed as meningitis, right temporosphenoidal abscess being suspected, but the possibility of a contralateral brain abscess was not seriously considered. Postmortem examination showed generalized edema of the brain, and the following abscesses: (1) a large subdural abscess; (2) a left frontal lobe abscess, and (3) a right temporal lobe abscess.

Collins was unable to find any reference to a chronic otitis media causing metastatic abscesses in the brain without showing any other evidence of generalized pyemia.

Frontal Lobe Abscess of Otitic Origin.—In two cases reported by van Caneghem,[58] an otitic infection was followed by an abscess in the frontal lobe; the abscess resulted from retrograde thrombophlebitis from the ear. In both cases, the infection passed from the outer part of the superior petrosal sinus to the inferolateral cerebral vein, the interior half of the superior petrosal sinus remaining intact. The sinus seemed especially narrow at the antero-internal segment, which van Caneghem thought prevented infection of the cavernous sinus.

Authorities are not agreed as to the pathogenesis of otitic cerebral abscesses. I believe that the most frequent type of "intracerebral abscess without macroscopically visible cortical invasion" is retrograde thrombophlebitis of a cerebral vessel. In my opinion, intracerebral abscess with a peduncle is a late stage of intracerebral abscess. The pedicle develops after the formation of the abscess cavity through a progression of the necrosis from the cavity of the abscess toward the cortex, thus following (but in an inverse direction) the route primarily taken by the causative thrombophlebitis. Van Caneghem thinks that in abscess from a retrograde thrombophlebitis, distinction should be made between a primary pedicle (furnished by the thrombotic vein which has carried the infection into the cerebral substance) and a secondary pedicle (which results from the progression of the abscess toward the cortex). In his first case there was evidence of a secondary pedicle because of the presence of a hard fibrous strand. The practical lesson to be drawn from both cases is the necessity for complete exposure of the dura of both cerebral fossae, in the presence of any infection of the meninges with the possibility of an abscess of the frontal lobe.

Temporosphenoidal Abscess with Cerebellar Symptoms.—A Hungarian author, Várady-Szabó,[59] described a case of temporal lobe abscess

58. van Caneghem, D.: Abscess of the Temporal Lobe Derived from an Otitic Infection of the Posterior Cerebral Fossa, Ann. d. mal. de l'oreille, du larynx **49**:340 (April) 1930.

59. Várady-Szabó, N.: Contributions to the Knowledge of Otogenic Brain Abscesses, Orvosi hetil. **73**:647 (July 6) 1929; abstr., Zentralbl. f. Hals-, Nasen- u. Ohrenh. **14**:710, 1930.

with persistent cerebellar symptoms: horizontal nystagmus toward the affected side, past-pointing, weakened tonus and rigidity at the nape of the neck without a Kernig sign (Brock's symptom). A temporal lobe abscess, the size of a nut, was incised.

Brain Abscess with Healed Middle Ear Suppuration.—Schmiegelow [60] observed five cases of otogenic brain abscess manifesting themselves when the primary middle ear suppuration had run its course and the tympanic membrane had healed. He compiled seventeen cases from the literature of the last fifty years. They generally occur in acute middle ear inflammations; much more rarely in chronic middle ear suppurations.

Recurrent Attacks of Meningitis from a Brain Abscess.—Beyer [61] reported a case of brain abscess with circumscribed purulent meningitis, originating from cholesteatomatous suppuration with Proteus infection. The abscess was operated on and healed with the formation of a hernia, which gradually became epithelialized. The tubal suppuration persisted, and after fourteen months led to an infection of the cicatrix, so that a new brain abscess appeared, which in its turn healed after evacuation by operation. At the end of half a year, there was a sudden onset of spastic states, which subsided again in a few days.

Latent Brain Abscess Revealed by Acute Meningeal Symptoms.—In this case,[62] meningitic symptoms appeared four days after a radical operation in which cholesteatoma was found. Lumbar puncture liberated turbid fluid containing streptococci. Enlargement of the cavity made at the radical operation revealed a large temporal lobe abscess. Repeated lumbar puncture, with gauze packing, led to a cure.

Latent Brain Abscess Discovered at Sinus Operation.—In Munyo's [63] case, on operation for lateral sinusitis, a thrombus was withdrawn from the sinus and a fistula discovered in the tegmen tympani, which led into a temporal lobe abscess. The patient recovered.

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60. Schmiegelow, E.: Otogenic Brain Abscess in Patients with Intact Tympanic Membrane or Cured Middle Ear Suppuration, Hospitalstid. **72**:260 (March 7) 1929; Cases of Otogenic Brain Abscess with a Normal Tympanic Membrane or with Healed Middle Ear Inflammation, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:273 (Aug.) 1929.

61. Beyer, H.: Recurrent Brain Abscess with Recovery, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:30 (Aug.) 1929.

62. Peyser, A.: Brain Abscess, Meningitis, Recovery (Demonstration of Patient), Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **18**:327, 1929.

63. Munyo, J. C.: Healed Otitic Brain Abscess, Arch. latino-am. de pediat. **23**:293 (May) 1929; abstr., Zentralbl. f. Hals-, Nasen- u. Ohrenh. **14**:509, 1929-1930.

Rupture of Abscess into Ventricle with Recovery.—Kovacs [64] said that so far as he knew his case was the third case of otogenous brain abscess with rupture into the ventricle, with recovery, to be reported.

SUBDURAL SPACE ABSCESS

Ruedi,[65] in his clinical and anatomic study, discusses (1) the anatomy of the cerebral meninges and (2) the literature on subdural abscess. Five cases are recorded in which a subdural suppuration was of otogenic origin. In three instances the infection was by contact of diseased bone with the dura. In one case, the subdural space had undergone a hematogenous infection from metastasis. A subdural abscess becomes encapsulated through agglutinations and adhesions of connective tissue. The arachnoid reacts by a toxic, exudative, productive inflammation. The brain is unaffected. Clinically, these cases run their course undiscovered, frequently because of the symptoms of other complications that dominate the picture, such as labyrinthitis, leptomeningitis or sepsis. When uncomplicated, subdural space abscess has an imperfectly marked but still a peculiar symptom-complex. Subdural suppuration is suggested by an onset marked by signs of meningitis, with a turbid sterile cerebrospinal fluid under high pressure, and the picture of a concomitant serous leptomeningitis. In addition, there may be focal symptoms from compression of the cerebral cortex. Both the meningitic and the focal symptoms show pronounced fluctuations. Subdural abscesses situated over the convexity of the brain offer the poorest prospects for recovery, on account of their usually wide extent. The following cases are reported:

Case 1.—A child died of diffuse leptomeningitis four days after a sinus operation; the subdural suppuration was discovered at the autopsy.

Case 2.—A girl of 17 years with bilateral ear suppuration of several years' duration, had a radical operation which was followed by diffuse labyrinthitis. A diagnosis of subdural suppuration was made. The patient died from leptomeningitis, which developed within ten hours as a sequel of a labyrinthine operation.

Case 3.—A woman, 23 years old, had left-sided chronic otitis media with cholesteatoma; radical operation was performed, and later operation on the labyrinth because of diffuse labyrinthitis. Puncture of the posterior cranial fossa gave negative results. Death occurred two days after the operation. Suppurative internal pachymeningitis over the left cerebral hemisphere was discovered at the autopsy.

Case 4.—In a case of subdural space abscess from acute middle ear suppuration, the subdural suppuration was not in the neighborhood of the bony focus but was

64. Kovacs, Arthur: A Case of Recovery from an Otogenous Brain Abscess After Rupture into the Lateral Cerebral Ventricle, Laryngoscope **38**:813 (Dec.) 1929.

65. Ruedi, L.: Otogenic Subdural Abscess, Monatschr. f. Ohrenh. **64**:3 (Jan.) 1930.

situated over the left convexity of the temporal and frontal brain, a great distance away.

CASE 5.—A boy of 15 years had a history of repeated attacks of middle ear inflammation. The evacuation of an extradural abscess revealed a local defect from which pus escaped. However, most of the pus was removed from the primary focus and consequently was not fully evacuated after a dural incision.

Zimanyi's [66] patient, aside from headache, had an absence of all general or local symptoms despite the long duration of an extensive subdural suppuration. There were multiple cavities, which were connected by narrow subdural tracts and could be traced only with difficulty. There were active granulations on the pia.

In the case observed by Hens,[67] the diagnosis was not made or the extensive subdural abscess discovered until the autopsy. The pathologico-anatomic findings showed a large subdural abscess over the right cerebral hemisphere without connection with the original pus focus in the ear. The author suggested that as the suppuration advances extradurally the pus becomes compressed between the bone and the dura. Resistance is offered, on the one hand, by the dura; on the other, by the bone. The abscess thus reaches a pacchionian granulation, which means a "weak" place in the dura, and the suppuration may thus enter the subdural space. After the invasion, the dura and the arachnoid endothelium become agglutinated and the abscess is thus completely closed off. As soon as the abscess, which was originally situated between the bone and the dura, has invaded the subdural space, the path which was traversed is no longer recognizable.

Cerebellar Abscess.—Generally speaking, one third of the cases of cerebellar abscess are preceded by lateral sinus infection.[68] Another third follow a suppurative labyrinth with complete destruction of the membranous labyrinth. The remaining third of the cases are the result of infection from the mastoid antrum.

Paralysis of Respiration.—Berggren,[69] calling at the hospital to see a patient, found him apparently dead from paralysis of respiration. Artificial respiration, massage of the heart and various stimulants were applied without avail. As respiratory paralysis is much more common in cerebellar than in cerebral abscess, the author exposed the cerebellar dura behind the sinus. Exploratory puncture produced thick grayish-brown pus. At the end of five minutes, the first spontaneous respira-

66. Zimanyi, V.: Subdural Abscess of Unusual Extent, Orvosi hetil. **74**:1143, 1930.

67. Hens, E.: Subdural Abscesses in Middle Ear Inflammations, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **19**:143 (Jan.) 1930.

68. O'Shea, V. H.: The Diagnosis of Brain Abscess, Brit. M. J. **2**:175 (Aug. 2) 1930.

69. Berggren, E. G.: Abscess of the Cerebellum, Complicated by Respiratory Paralysis: Recovery, Acta oto-laryng. **15**:101, 1931.

tion occurred, but of the Cheyne-Stokes type. The duration of the respiratory arrest was calculated as not less than thirty-five minutes. The patient gradually recovered.

Late Meningitis from Cerebellar Abscess.—Specht [70] reported a case of right-sided cerebellar abscess in which, after successful operation, there was a recurrence half a year later, which led to a fatal meningitis. For several prolonged periods the abscess was absolutely latent. At all times there was absence of cerebellar symptoms, in spite of the large size of the abscess. The author concluded that no explanation is possible.

BRAIN ABSCESS FROM INFECTIONS OF THE ACCESSORY NASAL SINUSES

Several avenues enter into consideration in the origin of rhinogenic abscesses.[71] In the fulminating cases, which are relatively rare, invasion occurs by the blood and lymph route; in the more frequent, protracted cases, by perforations of the bony posterior wall; also congenital osseous defects or osteomyelitis as a sequel of surgical intervention are especially apt to lead to brain abscess.

As a rule, symptoms due to the suppuration and to the increased intracranial pressure are present, but no focal symptoms. However, psychic disturbances, such as apathy or a pathologic euphoria, may be present; when the abscess extends backward, focal symptoms become demonstrable, such as speech disturbances and facial or motor paralysis. In the Basel Oto-Laryngological Clinic, since its establishment in 1897, a rhinogenic frontal brain abscess has been observed only twice.

CASE 1.—Chronic inflammation of both maxillary and frontal sinuses was followed by epidural abscess and right-sided frontal brain abscess. The brain abscess was not disclosed by cerebral puncture. Autopsy revealed a right frontal abscess, about the size of a pigeon's egg.

CASE 2.—The patient's illness was a sequel of influenza. There were severe frontal headache on the left side and rise of temperature, but without an increased nasal secretion. A few days later, swelling of the left side of the forehead occurred. Pus was voided, and the condition improved for about three weeks. Then the left-sided frontal pain returned, with fever of an irregular type; swelling of the left side of the forehead and the left upper eyelid occurred. Rhinoscopy showed no pathologic change. Radical Killian's operation disclosed that the posterior wall was eroded with exposure of a slightly granulating and distinctly pulsating dura. After a brief improvement, the patient complained of severe pain in the entire head; there was temporary somnolence. Ophthalmologic examination now revealed the presence of right-sided optic neuritis and an incipient choked disk on the left

70. Specht, F.: Contribution to the Question of Cerebellar Abscesses Without Cerebellar Symptoms, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **120**:23 (Jan.) 1929.

71. Friedmann, J.: Brain Abscess Following Frontal Sinus Suppuration, Schweiz. med. Wchnschr. **60**:240, 1930.

side. A second operation showed that the dura was now prominent at the side of the bony defect and had ceased to pulsate. On incision, a brain abscess was reached at 0.5 cm. It extended about 6 cm. backward and downward and in a mesial direction. About 50 to 70 cc. of fetid pus was voided. Following the operation the boy had no headache and was fully conscious. The decrease of intracranial pressure was very manifest in the increased frequency of the pulse; the rate rose from 68 to 112. Meningitis and death occurred seven weeks after the onset. Autopsy showed purulent meningitis, brain abscess and general infection, with softening and swelling of the spleen and cloudy swelling of the liver and kidneys.

Comment: It is of interest that after the exposure of the extradural abscess, the dura still pulsated, in spite of the presence of a brain abscess. This has been my experience in several instances.

Frontal Lobe Abscess from the Frontal Sinus.—In 1922, I collected 150 cases of frontal lobe abscess, 64 of which took their origin from the frontal sinus. Skillern and Coates [72] have augmented these by 27, so that to date there are 91 cases, which is about one half of the total 177 frontal lobe abscesses reported.

They review in detail thirty cases of frontal lobe abscess from the literature, in which the patients have been reported as recovering after operation. They stated that in reviewing the treatment, it seems to them great stress is laid on certain procedures which appear quite unnecessary, while other measures of the utmost importance are merely mentioned or ignored.

In 1922 I stated that at least 45 per cent of frontal lobe abscesses were diagnosed only on the autopsy table. Some improvement has occurred, but it is only within the past two years that expert neurologic examinations have played an important rôle.

In most of the cases that have been reported in which a fistula has formed, the patient has recovered. Skillern and Coates ask in operating, "why not emulate nature by making a fistula of sufficient size for adequate drainage, and keep it patulous by packing until the abscess cavity is closed by slowly forming granulation tissue?" They have found a clean field in such cases to be unnecessary. It was previously thought that the high mortality was due to exposure of the meninges to purulent secretion with resulting meningitis. This has not proved to be so, for if the patient survives the operation, the cause of death is usually a secondary abscess.

The first intracranial dressing should be left *in situ* ten days or two weeks; the iodoform gauze is slowly removed on succeeding days, only that which is loose and does not adhere to the brain substance being taken away. The patient should be confined to bed at least five or six weeks.

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72. Skillern, R. H., and Coates, G. M.: An Enormous Frontal Lobe Abscess Following Infection of the Frontal Sinus: Operation; Recovery, Ann. Otol., Rhin. & Laryng. **39**:398 (June) 1930.

Later Death from Healed Frontal Lobe Abscess.—Mayer [73] described a case of frontal brain abscess after acute frontal sinus suppuration, which had been presented by him as healed in 1918. After having felt well, the patient suddenly died with tonic spasms three months later, following an illness of but one day. Autopsy showed a healed abscess, with a small cicatrix between the frontal brain and the dura. There were cerebral edema and status lymphaticus. It is doubtful whether the cause of death was the brain disease or the status lymphaticus. "The frontal lobe of the brain makes rather wide excursions with respiration, therefore, in the presence of cicatrices of the frontal lobe, the pulsatory and respiratory movements stimulate the brain cortex, which often results in vertigo, vomiting and convulsions after healing."

TREATMENT OF BRAIN ABSCESS

In the absence of localizing symptoms, MacCuen Smith [74] thinks that the temporal lobe on the same side as the aural lesion should first be explored. Following failure to locate the pus, the cerebellum of the same side should be punctured. Most temporosphenoidal abscesses are caused by direct extension from the site of the disease. "This is the reason why the surgeon should follow the avenue of infection. It also provides for very much better drainage than an opening made higher up." Even *in extremis,* intervention is called for, since the symptomatology may be due to intracranial pressure alone, which evacuation will relieve.

Uffenorde [75] fears the associated progressive encephalitis with rupture into the ventricle that follows a reaccumulation of pus. Consequently, control of the multilocular abscess cavity is imperative. This he thinks can be done by wide opening of the abscess with drainage by glass tubes.

Cerebral Hernias Following Brain Abscess.—Fremel [76] recognized two factors: (1) a bone-dura defect, and (2) internal pressure. The latter is caused by encephalitis or increased cerebrospinal fluid. In the presence of encephalitis, a dura that has been merely punctured may break down, thus permitting prolapse of the brain. Small abscesses and well encapsulated ones seldom cause prolapse. Prolapse occurs when there is some condition in addition to the abscess. It is usually

73. Mayer, O.: Sudden Death After Healed Frontal Brain Abscess, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **14**:653, 1930.

74. Smith, S. MacCuen: Otitic Brain Abscess: Diagnosis and Treatment, M. Times, New York **57**:276 (Oct.) 1929.

75. Uffenorde, W.: Treatment and Prognosis of Otogenic Brain Abscess, Klin. Wchnschr. **9**:1004, 1930.

76. Fremel, F.: Postoperative Brain Prolapse, Arch. Otolaryng. **11**:238 (Feb.) 1930.

encephalitis; less often it is pus retention or another abscess. Fremel, to obviate as far as possible the danger of prolapse, uses a needle for exploration instead of a knife. In the Neumann clinic, prolapse occurred in fourteen out of thirty-nine cases of cerebral abscess.

Iodized Sesame Oil Visible One Year After Abscess Had Healed.—In Traina's [77] case of healed temporosphenoidal lobe abscess, iodized sesame oil, 40 per cent, that had been injected was visible after a year, during which time the patient was free from all disturbances.

OSTEOMYELITIS OF THE SKULL

Osteomyelitis from Vincent's Spirillum.—A case of sinusitis and fatal meningitis due to Vincent's spirillum is reported by Seecof [78] in which death occurred four days after the onset of symptoms. Necropsy revealed chronic infection of the frontal, ethmoid and sphenoid sinuses; osteomyelitis of the frontal bone; cellulitis of the left orbit; localized meningitis, and pulmonary gangrene. In all these locations, Vincent's organisms were present in association with other bacteria. The intracranial lesions appear to have resulted from the spreading of the infection through the diploe of the frontal bone; the pulmonary lesion, from aspiration from the throat.

Obscure Osteomyelitis of the Sphenoid.—The following case was reported by Myers: [79]

> On Dec. 31, 1929, the patient was hospitalized for twenty-eight hours for the fitting of a belt to the back and for a tonsillectomy; the latter procedure appears to have been merely an adjunct to other treatment. A tag of tonsil was removed on February 14.
>
> On February 26, he complained of pain in the lower part of the back, of headaches indefinitely distributed, and of dizziness; he was irascible, excitable and restless.
>
> On February 27, he was again excitable and irascible, and had a mild headache with dizziness, but complained principally of the pain in the lower part of the back. He could not swallow fluids. From time to time there were violent contractions of the abdominal muscles, drawing of the lower extremities upward and of the body forward. The fauces showed recent enucleation of the tonsils and more redness and congestion than should have been expected so long after the tonsillectomy.
>
> At autopsy there was a definite and marked osteomyelitis of the occipital, sphenoid and petrous portion of the temporal bone on the left, with evidence of

77. Traina, S.: Serous Collection in the Left Temporosphenoidal Lobe, Following upon a Regional Abscess, Valsalva **5**:475, 1929.

78. Seecof, D. P.: Sinusitis and Fatal Meningitis Due to Vincent's Spirillum, Arch. Otolaryng. **10**:384 (Oct.) 1929; abstr., J. A. M. A. **93**:1681 (Nov. 23) 1929.

79. Myers, Glenn: Osteomyelitis of the Skull, J. A. M. A. **95**:406 (Aug. 9) 1930.

septic meningitis confined to the base of the skull, and especially in relation to the area of osteomyelitis.

Obscure Osteomyelitis of the Sphenoid as the Cause of Cavernous Sinus Disease.—In the first case reported by Uffenorde,[80] an acute unilateral inflammation of the accessory nasal sinuses was followed by sepsis. A woman, aged 44, suddenly developed chills and severe pains in the occiput, in the trigeminus and in the teeth. She died after one week. Necropsy showed that the infection had commenced in the sphenoidal sinus, perforated into the posterior orbital cavity, caused thrombosis of the ophthalmic vein and of the cavernous sinus, and then led to meningitis. The fourth case developed in a puerpera, aged 20. At first, eclampsia was considered, but attacks of jacksonian epilepsy were inconsistent with such a diagnosis. Necropsy revealed meningitis from suppuration of the frontal sinus and of the ethmoid bone. Osteomyelitis in the frontal bone had led to perforation into the cranial cavity.

Temporosphenoidal Lobe Abscess from the Frontal Bone.[81]—An infection of the left frontal sinus extended to the wall of the sinus, and involved a large part of the frontal bone, with the formation of an abscess in the right temporal lobe. The patient recovered.

THE NOSE AS AN AVENUE OF INFECTION IN CEREBRAL COMPLICATIONS

Clark's [82] specimens, prepared from animals, indicate that a solution of potassium ferrocyanide and iron ammonium citrate, which had simply been dropped into the nasal cavities, was very rapidly absorbed by the nasal mucous membrane and, within one hour, had reached the surface of the brain. The intermingling of the prussian blue granules with the cells of the olfactory epithelium is evidence that the solution had penetrated throughout the thickness of the epithelium. This absorptive power may well be correlated with special properties of the olfactory mucosa (which enables the sensory epithelium to be brought into intimate contact with substances of widely differing chemical composition known as odoriferous).

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80. Uffenorde, W.: Suppuration of Accessory Nasal Sinuses, Deutsche med. Wchnschr. **56**:776 (May 9) 1930; abstr., J. A. M. A. **95**:377 (Aug. 2) 1930.

81. Mason, J. M.: Abscess of the Right Frontal Lobe of the Brain from Osteomyelitis of the Frontal Bone Originating in the Left Frontal Sinus, S. Clin. North America **10**:889 (Aug.) 1930.

82. Clark, W. E. le Gross: Report of the Committee on Vaccination on an Anatomical Investigation into the Routes by Which Infections May Pass from the Nasal Cavities into the Brain, Reports on Public Health and Medical Subjects, no. 54, Ministry of Health, Paper 15, London, His Majesty's Stationery Office, 1929.

Yates found evidence of nasal infection in thirty-four cases of epidemic encephalitis. It was accompanied by well marked ciliary paralysis and apparently by increased power of absorption in the mucosa. He suggested that this heightened power of absorption might well be a factor in permitting the entrance of a filter-passing virus.

Clark thinks that it is possible to postulate a continuous flow of the absorbed fluid along the olfactory nerve fibers from the epithelial layer up to the brain. This conception is in harmony with the recent work on the development of the pia-arachnoid. Harvey and Burr [83] drew the conclusion that in Amblystoma embryos (and presumably in vertebrates generally) the leptomeninges arise from the cells of the neural crest; in other words, the olfactory epithelial cells, together with their proximal processes, the olfactory nerve fibers and their neurilemmae, have the same origin as the prolongations of the pia-arachnoid which appear to accompany them. It may be affirmed that the main route by which the absorbed fluid in these experimental rabbits reached the surface of the brain is provided by the perineural sheath spaces.

In a few cases, minute capillaries, which ran in among the fibers of an olfactory nerve bundle and pass through the cribriform plate, were found to contain prussian blue granules, and occasionally a larger vessel, presumably a small vein, also passing through the cribriform plate but along the side of a nerve bundle, was found with endothelial lining. It is evident that these vessels will provide theoretical routes along which infections of a pyogenic nature, accompanied by the formation of thromboses, may pass from the nasal into the intracranial cavity. Hajek emphasized the rôle of these perforating veins in rhinogenous brain complications.

The blood vessels which run through the cribriform plate actually lie in the sheaths of the nerves that they accompany, that is to say, among the meshes of the trabecular tissue, which is but an extension of the pia-arachnoid through the cribriform plate into the nose.

The nasal mucosa is linked up directly with the substance of the brain by the protoplasmic axis cylinders of the olfactory nerves and of the trigeminal nerve, thus connecting the mucous membrane with the olfactory bulb and the medulla.

Clark's conclusion is that the herpetic virus reaches the central nervous system through the medium of the nerves supplying the peripheral areas inoculated. Thus inoculations of the scarified cornea of rabbits led to lesions in the pons whither the infection has been carried by the ophthalmic division of the trigeminal nerve, a conclusion that has also

83. Harvey, S. C., and Burr, H. S.: The Development of the Meninges, Arch. Neurol. & Psychiat. **15**:545 (May) 1926.

been reached by Marinesco and Draganesco.[84] Basing his arguments on the fact that the virus seems to grow within cells rather than in the tissue fluids, and that an acute herpetic lesion occurs at the central origin or terminus of a nerve along which the virus is conveyed, Goodpasture concluded that the virus enters the axis cylinder of a peripheral nerve and grows within this protoplasmic filament until it reaches the body of the cell itself, which it destroys, the liberated virus then spreading to the surrounding tissues. He excluded the possibility of an ascending herpetic neuritis by a propagation of the virus through the cells of the neurilemma because of the absence of evidences of inflammation and of herpetic cell inclusions along the course of the nerve. Using inoculations of bacterial emulsions and injections of trypan-blue into peripheral nerves, Thiele and Embleton[85] concluded that the more soluble substances may pass directly up the axis cylinders to the spinal cord, that infection may travel along the perineural lymphatics but does not spread in this way directly to the cord or cerebrospinal fluid, and that therefore direct invasion to the cord itself is along the axis cylinders themselves. In their experiments, Orr and Rows[86] induced an ascending infection by placing celloidin capsules containing a broth culture of a micro-organism in contact with a peripheral nerve. They found evidence of ascending "lymph" stream to the central nervous system, the current of which lies in the spaces of the perineural sheath, and along this route toxins reach the spinal cord and brain and may spread to some degree in the "lymph" spaces of the pia-arachnoid. These pathologic investigations have an important bearing on the anatomic experiments reported in Clark's paper, in which is demonstrated the readiness with which a solution absorbed through the nasal mucosa may track up along the perineural sheaths of the olfactory nerves. It seems certain that these sheaths are of paramount importance in the spread of infective processes from the nose to the brain. Westenhoffer[87] believes that in cerebrospinal meningitis the infection of the meninges takes place from the sphenoidal air sinus, partly because *the initial meningitis is so constantly a basilar infection.* Embleton and Peters[88] suggested that the reason adults were less susceptible to this disease was because they show a diminished tendency to sphenoidal empyema. Turner and

84. Marinesco, G., and Draganesco, S.: Ann. Inst. Pasteur **37**:753 (Aug.) 1923.

85. Thiele and Embleton: Paths of Spread in Bacterial Infection, Proc. Roy. Soc. Med. (Sect. Path.) **7**:69, 1913-1914.

86. Orr, T. G., and Rows, R. G.: Toxi-Infection of the Central Nervous System, Edinburgh M. J. **17**:78 (Aug.) 1916.

87. Westenhoffer: Pathologische Anatomie und Infectionsweg bei der Teruchstarre, Berl. med. Wchnschr. **42**:737 (June 12) 1905.

88. Embleton and Peters: Cerebrospinal Fever and the Sphenoidal Sinus, Lancet **1**:1078 (May 22) 1915.

Reynolds [89] described a case of leptomeningitis following intranasal operation on the ethmoidal air cells, in which the perineural spaces of the branches of the olfactory nerves lying in the septal mucosa were dilated and invaded by polymorphonuclear leukocytes. In a similar case Modiowski [90] concluded that the infection passed up along the perineural spaces. Logan Turner [91] quoted a case reported by Hayen. A man had an attack of epistaxis and sought the aid of a pharmaceutical chemist, who plugged his nose with a tampon soaked in perchloride of iron. A few days later the man died from meningitis. At the necropsy it was found that the olfactory bulbs were stained brown and gave the prussian blue reaction.

Turner and Reynolds [92] devote the eighth of their most valuable contributions to an understanding of the paths of infection to the brain, meninges and venous blood sinuses from the ear and nose, by reporting in detail the microscopic findings in a case of chronic suppuration in the ethmoid and sphenoid air sinuses which was followed by acute leptomeningitis.

The patient had had an intranasal operation two years previously, but microscopically there was no evidence of fracture. The infection entered the brain by way of a perineural space surrounding one of the branches of the olfactory nerve.

Rhinorrhea Continuing for Years.—Bulson and Bulson [93] reported a case in which rhinorrhea continued for years.

A married woman, aged 40, was seen first in February, 1921. At that time the turbinates were fairly normal.

On Sept. 12, 1922, she reported that two months previously a physician had operated on her nose, removing the turbinates from both sides, since which time she had been in bed almost continually, suffering from severe headaches, general weakness and a constant flow of clear watery fluid from the right side of the nose. The fluid was cerebrospinal fluid. The polypoid material in the region of the anterior ethmoidal cells was removed and the entire upper naris was swabbed gently with a 5 per cent alcoholic mercurochrome solution. She was given a 25 per cent solution of mild silver protein to use in the nose with a medicine dropper while the head was tipped back, and advised not to use a douche or atomizer and not to blow the nose violently.

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89. Turner, A. L., and Reynolds, F. E.: J. Laryng. & Otol. **41**:73 (Feb.); 442 (July); 717 (Nov.) 1926; **42**:525 (Nov.) 1927; **43**:34 (Jan.) 1928.

90. Modiowski: Verhandl. d. internat. Laryng. Kong., Berlin, 1911.

91. Turner, A. L.: The Spread of Bacterial Infection from the Nasal and Pharyngeal Cavities by Way of Lymphatic Channels, Edinburgh M. J. **7**:409, 1911; Verhandl. d. Internat. Laryngo-Rhin., Kong., Berlin, 1911, pt. 3, p. 65.

92. Turner, A. Logan, and Reynolds, F. E.: A Study of the Paths of Infection to the Brain, J. Laryng. & Otol. **44**:801 (Dec.) 1929.

93. Bulson, Albert E., and Bulson, Eugene, L.: Cerebrospinal Rhinorrhea Following Intranasal Surgery, J. A. M. A. **93**:1969 and 1970 (Dec. 21) 1929.

She again remained in bed almost continuously for a period of three months, during which time she was physically unable to be up and around, and throughout the entire time suffered from headaches, and from a continuous dripping of the cerebrospinal fluid.

Late in 1924 the dripping from the nose was less profuse. There also was less headache, and she had gained in strength and weight. Examination showed that there was considerable granulation tissue in the region of the posterior ethmoid. There was no discoverable pulsation. As late as June 25, 1928, practically six years after the nasal operation, a specimen of fluid from the nose showed it to be cerebrospinal fluid. The fluid appeared periodically and sometimes for weeks there would be little if any dripping from the nostril.

Calcifications in Relation to Sinusitis.—Recently there has been noted [94] a definite coincidence between the occurrence of calcification and the presence of infection of the sinuses. The atypical neuralgias are frequently associated with meningeal calcification.

There is little presented in the literature to indicate a pathologic relationship between meningeal calcification and a pathologic process in the sinuses. Hirtz reported a series of 144 cases studied in relationship to the pineal gland, and of the series, 56 per cent revealed calcification; but calcification was not found without associated evidence of deep perisinusitis. The falx is the most common site, with the areas in the dura beside the superior longitudinal sinus, and the tentorium next in frequency. Congestion of the vessels within the involved area is usually present. Adhesions are frequently observed. Weber has stated that ossification of the dura does not belong to genuine tumors but to inflammations. The author believes that these osteitic changes are a defense reaction caused by the deep perisinuous disease.

Babbitt observed six cases, all of which presented striking phases of neuralgic pain complex. All presented intranasal pathologic processes of a degree justifying surgical intervention. Of these six, four presented calcification at the base of the brain around the pituitary area, and two, the same condition about the longitudinal sinus.

Pathology of Rhinorrhea.—In 1929, I [95] reported three cases of ingrowth of the mucous membrane from the nasal cavity into the arachnoid and cerebral tissue, with postmortem observations. Taken together, these three cases explain rhinorrhea of traumatic or spontaneous origin as arising through the development of a fistulous traumatic or olfactory perineural tract by the ingrowth of low grade mucous membrane, combined with proliferation of the arachnoid when the two

94. Babbitt, James A.: The Coincidence of Meningeal Calcification and Nasal Pathology, Ann. Otol., Rhin. & Laryng. **38**:320 (June) 1929; abstr., Arch. Otolaryng. **10**:543 (Nov.) 1929.

95. Eagleton, Wells P.: The Cerebrospinal Fluid as an Aid to Diagnosis and Treatment of Inflammatory Diseases, J. Laryng. & Otol. **44**:721 (Nov.) 1929.

membranes are approximated. Rapid epithelial ingrowth occurs because of the adhesion of the arachnoid to the dura at the site of the cleft. The membranous growth into the defect prevents the bony or fibrous union, which is normally slow in all flat bones, because of lack of motion to stimulate callous formation. The persistence of the flow of cerebrospinal fluid converts the tract into a permanent endothelial-lined channel. Continuous abnormal brain pressure, by enlarging an originally small bony opening, explains the increase by absorption of the bone of the cribriform openings which conduct the perineural arachnoid prolongation from the olfactory bulb, as recorded in cases of cerebral tumor with rhinorrhea. Through the enlarged perineural or traumatic tract the arachnoid lake and the nasal mucous membrane become continuous in cases in which the tract enters the chiasmal cisterna (tumor or operative fracture), or when it forms an intracerebral cyst, which may communicate with the ventricle in cases of traumatic cerebral laceration. Sudden fulminating meningitis, and rapid termination from dissemination of the infected fluid through the sylvian fissure meshes to the cortex and basal cisterna, occur in all cases of rhinorrhea when limitation is unsuccessful.

Obscure Sphenoid Sinus Suppuration in Relation to Intracranial Complication.—There is apparently some relationship between glaucoma and suppurative disease of the sinuses; I have seen its occurrence, and one of Kramer's [96] patients was admitted for glaucoma. In my case,[97] chronic glaucoma actually did develop two years after cure by operation for meningitis resulting from suppuration of the frontal and ethmoid sinuses.

Other cases reported by Kramer follow:

CASE 2.—In this case death followed a pneumococcic meningitis in two days. At postmortem examination the meningitis was found to originate from sphenoidal suppuration. There was an osteomyelitis of the sphenoidal bone, apparently of several weeks' or months' duration, but without any cerebral symptoms, although the patient complained of a cold in her chest for several weeks.

CASE 3.—The patient had been ill for four months with headache, pains in the arms and legs and progressive weakness. She vomited for two weeks before admission, at which time she had symptoms of meningitis, lumbar puncture showing 1,500 cells, with 70 per cent polymorphonuclears. Death occurred a few days after spheno-ethmoidectomy, which showed Friedländer's bacillus (the so-called ozena group).

Postmortem examination revealed purulent meningitis and encephalitis, the left sphenoid contained a creamy, yellow pus; the right sphenoid, a small amount of pus, as did the ethmoids on both sides. The occipital bases were soft and friable,

96. Kramer, Rudolph: Intracranial Complications Following Sphenoid Infection, Laryngoscope **39**:573 (Sept.) 1929.

97. Eagleton, Wells P.: The Philosophy of Meningitis, Laryngoscope **39**:627 (Oct.) 1929.

with a path of necrotic bone leading from the left sphenoid sinus through the basi-occiput into the cerebellar fossa. The dura was perforated. The pia was adherent over the perforated dura. Culture revealed atypical Friedländer's bacillus.

CASE 4.—Autopsy presented a brain abscess of sphenoid sinus origin. Despite its enormous size there were no focal symptoms of abscess, but the patient presented signs of a mild spheno-ethmoiditis. She was thought to have a hemorrhagic nephritis. Kramer said that there is only one other case in the literature.

CASE 5.—For four or five months the patient suffered from vomiting and later from pain in the neck, with a septic temperature. Finally headache, coma and a discharging ear developed.[98]

Postmortem examination showed a sinus tract of 1.5 cm. in the body of the sphenoid. It ran from the roof of the nasopharynx through the sphenoid bone. The sella turcica was eroded and the periosteum of the base was thickened. There were basilar meningitis, hypophysitis and ependymitis.

CASE 6.—This case is an example of blood stream infection associated with meningitis of sphenoid origin.

An infection of the upper respiratory tract of one week's duration was followed by headache, restlessness, and impaired hearing, with the ear drum full and red. On admission, the patient was thought to be suffering from otitic meningitis. Lumbar puncture and blood cultures gave pneumococcus, type III.

Postmortem examination revealed purulent meningitis, empyema of the sphenoid, parenchymatous degeneration of the heart, liver and kidneys, thrombosis of the left external iliac vein, pulmonary embolus, and multiple infarcts.

The meningitis was of the fulminating type following sphenoid sinus suppuration, the first symptoms of meningeal irritation appearing two days before death. The infection of the upper respiratory tract, which was the apparent cause of the sphenoid involvement, started five days before. The pathway of infection from the sphenoid sinus to the meninges was not evident macroscopically. "One may hazard the assumption that the meninges were infected by way of the perforating vessels in the sphenoid sinus walls."

From operations and autopsies in such cases, I [99] know that osteomyelitis of the sphenoid is accompanied by headache, either frontal or occipital, or both, by some stiffness of the neck, and by occasional vomiting. These symptoms are also seen in an occasional uncomplicated sphenoid sinusitis or in basilar meningitis. They seem to me to be a good working basis for establishing the diagnosis of and the treatment for osteomyelitis of the sphenoid before serious, perhaps irremediable, intracranial complications have risen. The persistence of mild meningitic signs in addition to the foregoing (sinusitis, headache, vomiting, stiffness in the neck), and the finding in the spinal

98. Eagleton, Wells P.: Case of Recrudesence of a Patent Infection (Streptococcia) in the Marrow Substance of the Petrous Apex and the Sphenoidal Sinus: Immediate Blood Stream Infection; Diffuse Thrombophlebitis of Cerebral Veins; Basilar Meningitis; Cerebral Compression from Encephalitis, Edema of Brain, and Nonobstructive Internal Hydrocephalis, in press.

99. Eagleton, Wells P.: Suppurative Meningitis of Otitic and Nasal Origin: Its Relation to Blood Stream Invasion of the Pial Vessels, Arch. Otolaryng. **15:** 885 (June) 1932.

fluid of indications of infection, are definite evidences of an intracranial complication probably of sphenoidal origin.

The ears often are a cause of confusion. This is of frequent occurrence in cases of sphenoidal origin, as evidenced by Burger, who, in thirteen collected cases of meningitis of sphenoidal origin, found that otologic operations had been performed on six. The origin of the meningitis was not discovered until postmortem examination. Kramer emphasized the lesson that meningitis may be of long standing, that the symptoms may be vague and easily overlooked, that they may simulate a brain abscess by their mildness and chronicity and that brain abscess and cavernous sinus thrombosis may be entirely symptomless in the presence of other lesions.

Druss [100] reported two cases of intracranial complications associated with disease of the sphenoid bone.

Case 1.—A woman had a history of intermittent vomiting for four or five months. A month previous she experienced severe pain in the back of the head and had a profuse nasal discharge, followed by an irregular temperature and extreme weakness. Three days prior to admission, she became unconscious.

When admitted to the hospital there were a grayish purulent exudate on both corneas, rigidity of the neck, and bilateral Kernig and Babinski signs. The deep reflexes were increased.

Lumbar puncture revealed a slightly cloudy yellow fluid under normal pressure. Twenty-seven cells (all lymphocytes) were found. Culture revealed Streptococcus gamma (a slow growing organism).

At autopsy pus was found in the ethmoid cells and in the left antrum. In the body of the sphenoid bone there was a defect, a sinus tract which extended from the roof of the nasal cavity into the body of the sphenoid for about 1.5 cm. The cavity contained cheese-like detritus. The sella turcica was eroded, and the dura over the sphenoid bone thickened and in places was lifted from the underlying bone. Purulent meningitis was fairly well circumscribed about the cerebellum, pons, medulla and midbrain.

The sinus tract apparently was an extension of infection from the nasopharynx. This produced an osteomyelitis, which had resulted in meningitis. The possibility of a rest of Rathke's pouch (craniopharyngeal canal) which had become infected was considered. Sokoloff [101] stated that the craniopharyngeal canal is present in about 0.3 per cent of all adults and in about 10 per cent of new-born infants. The canal is usually obliterated about the third month of the embryo.

Case 2.—In this case there was an unusual complication of an abscess of the frontal lobe secondary to sphenoidal involvement. The patient, two months prior, experienced severe frontal headache, stuffiness in the nose, nausea, vomiting, poor

100. Druss, J. G.: Intracranial Complications Associated with Disease of the Sphenoid Bone, Arch. Otolaryng. **10**:39 (July) 1929.

101. Sokoloff, N.: Der Canalis craniopharyngeus, Arch. f. Anat. u. Physiol., 1904, p. 71.

vision, and generalized weakness. One week later, she had a chilly sensation followed by a temperature of 105 F., with pyuria and hematuria. About five weeks before admission, she complained of pains in the back of the neck. A spinal tap showed the cerebral fluid under increased pressure and containing 20 cells. On admission she appeared drowsy, but responded to all questions. The deep reflexes could not be elicited. The abdominal reflexes were sluggish. Bilateral amaurosis, bilateral papilledema with hemorrhages, fixed and dilated pupils, paresis of conjugated deviation on the left, paresis of the upward gaze and right facial weakness were present. Lumbar puncture showed eight cells.

A diagnosis of basilar meningo-encephalitis was made.

Autopsy showed that the brain was under increased tension. There were purulent sinusitis of the right sphenoid sinus, an abscess of the right frontal lobe and a basilar meningitis in the interpeduncular space and about the optic chiasm. The gyri recti of the frontal lobe presented a perforation which communicated with a cavity within the substance of the brain. The exudate extended back to the pituitary fossa, with a perforation of the bony plate forming the roof of the sphenoid sinus.

Microscopic examination of the sphenoid bone showed a mild inflammatory process in the left sphenoid sinus. The right sphenoid sinus contained a mucoid exudate. The mucous membrane on the posterior wall showed submucosal edema and infiltration with polymorphonuclear leukocytes and polypi.

There was a small cavity behind the right sphenoid sinus, with a perforation at the roof. This cavity was, in all probability, an accessory sphenoid sinus, as, on serial section, no definite communication could be established between it and the sphenoid sinus. The dura overlying the sphenoid was inflamed, as was the pituitary gland. No perforation or disease was noted in the roof of either sphenoid sinus.

SUPPURATIVE MENINGITIS OF AURAL AND NASAL ORIGIN

In the Ear Clinic of the Second State University in Moscow,[102] 1920 to 1928, 111 cases of meningitis of aural origin were observed. Cholesteatomata were encountered in 34 chronic cases, and labyrinthogenic meningeal inflammation in 25 cases (39.6 per cent). Cure was obtained in 17.1 per cent. Chronic cases showed a favorable outcome in 21.9 per cent; the acute, in 7.8 per cent. Homolateral facial paralysis was found in 20.7 per cent; in some cases, homolateral abducens paralysis and dilatation of the pupils were observed. Introduction of methenamine into the blood and by endolumbar injection had a beneficial effect. Iso-acetylhydrocupreine injection (vucin), in the author's patients, had no influence on the course or on the outcome of these leptomeningitides.

In fifty-one cases of otogenic meningitis, Gusic[103] had three cases in which cure was obtained (one chronic otitis with acute labyrinthitis and two cases of acute influenza otitis; labyrinthectomy was performed

102. Schpajer, E.: Statistics and Symptomatology of Leptomeningitis of Otogenic Origin, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **16**:104, 1930.

103. Gusic, B.: Three Cured Cases of Purulent Otogenic Meningitis, Otolaryng. slavic **1**:192, 1929; abstr., Zentralbl. f. Hals-, Nasen- u. Ohrenh. **14**:157, 1929.

in two). Repeated lumbar or suboccipital puncture, following the removal of the primary focus, yielded the best results. The puncture is followed by a subcutaneous injection. Large intravenous doses of serum are also recommended.

Pathologic Anatomy of Meningitis.—Davis [104] pointed out that otitic meningitis of the middle fossa is uncommon, because in this area the dura is not penetrated by nerves or vessels from the ear, except in children in whom the petrosquamous suture is still pervious. Otitic meningitis is usually basal. The suppuration is found in the basal cisternae and in the posterior fossa because of the passage of the nerves and vessels through the petrous bone. A general blood infection occurs in cases in which the path of infection is untraceable.

Infection by the internal ear rapidly spreads to the cisterna pontis, then to the cisterna basilis, overflowing along the cerebral vessels. The cisterna magna is the last of the cisternae to be infected, hence drainage by Haynes' operation, or any other method, is of little value.

The dura mater of the fossa subarcuata is pierced by a vessel that arises in the cancellous bone surrounding the semicircular canals and labyrinth. Also, cancellous bone is in direct continuity with the retrofacial mastoid cells and with the sinus tympani of the middle ear. Suppuration in this area may cause infection of the meninges, especially as retrofacial cells may escape notice during the mastoid operation.

Suppuration from the lateral sinus spreads to the posterior fossa, and an oyster-like abscess covers the under surface of the cerebellar hemisphere. It finally reaches the cisterna magna. When the path of infection is through the internal ear, translabyrinthine drainage by the internal auditory meatus and drainage through the inner wall of the mastoid antrum in front of the lateral sinus should be performed early.

When the path of infection is through the lateral sinus, the posterior fossa should be drained both in front of and behind the lateral sinus. When the cisternae are infected, drainage irrigation with Ringer's solution and the use of serum have been advocated (Jenkins and Eagleton), but Davis has not followed this method.

Labyrinthitis as the Cause of Meningitis.—Druss [105] gives the pathways of infection in labyrinthitis.

Extension along the aquaeductus cochleae offers direct communication between the subarchnoid space and the scala tympani of the basilar turn of the cochlea (perilymphatic).

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104. Davis, E. D. D.: Otitic Meningitis: Its Morbid Anatomy, Prevention and Early Diagnosis, J. Laryng. & Otol. **44**:249 (April) 1929.

105. Druss, J. G.: Pathways of Infection in Labyrinthitis: Report of Three Different Types, Arch. Otolaryng. **9**:392 (April) 1929.

The usual method of invasion of the labyrinth from the meninges is by way of the internal auditory meatus. Abscess formation in the internal auditory meatus is not infrequent, and with the pressure exerted on the internal auditory artery and vein, necrosis of the central part of the labyrinth may result. The periphery of the labyrinthine capsule is supplied by vessels of the dura which rarely anastomose with the small branches of the internal auditory artery (Siebenmann).

Druss reported the following case:

A 3 months' old child died twenty-four hours after admission to the hospital. Postmortem examination showed acute diffuse purulent meningitis, intraventricular hemorrhage in the brain, cerebral softening and hemorrhage. Microscopic examination of the temporal bone revealed a slight purulent exudate in the cavity of the middle ear and in the eustachian tube. A marked purulent and hemorrhagic infiltration was present in the perilymphatic spaces, particularly in the basal turn of the cochlea, in the scala tympani and vestibule. The ductus cochlearis and the other endolymphatic spaces contained serosanguineous exudate, but the cellular elements were not present. Hemorrhage was seen in the spiral ligament in the basilar and second turn of the cochlea. Considerable purulent hemorrhagic infiltration was present in the internal auditory meatus about the main blood vessel and nerves. The purulent exudate could be traced into the modiolus and Rosenthal's canal, making its entrance into the membranous labyrinth.

The fact that the child had congenital syphilis (Wassermann reaction was 4 plus) was taken into consideration. The hemorrhage in the brain and in the spiral ligament of the cochlea was probably a syphilitic manifestation.

Meningitis from a Healed Labyrinth, the Immediate Cause Being Exposure to Sunlight.—Theissing [106] contributed a description of the histologic picture in a case of late meningitis, developing as a sequel of a labyrinthitis which had apparently healed by new connective tissue and bone formation. It is of clinical interest that the meningitis followed an intensive exposure to sunlight, the etiologic significance of which has recently been emphasized by Strusberg.

The case leaves it an open question whether direct action of the rays was the immediate cause, or whether overheating caused a sudden exacerbation of the latent focus in the petrous bone, as an old labyrinthitis was the starting point of the infection.

The patient, thirteen months previously, suffered from acute otitis media. Three months later a radical operation was done. The operation was followed by severe persistent vertigo, which subsided. As the surgical wound failed to heal, frequent sunlight irradiation was applied, and eight days before admission the patient exposed herself to sunlight irradiation for five hours. This was followed by severe headache. On the next day, persistent vomiting occurred. At the time of admission there was a rather profuse secretion with a postauricular fistula. The right ear was totally deaf and calorically nonirritable. The cerebrospinal fluid

106. Theissing, G.: Bone Formation in the Labyrinthine Cavities After Labyrinth Destruction with Late Meningitis: A Contribution to the Question of Isolated Meningitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:451, 1930.

was turbid, with a positive Pandy reaction. The ocular fundus showed blurred papillary boundaries on the nasal side. As there was a possibility of infection of the meninges, the surgical cavity was freely exposed, showing granulations in the tympanic region. Postmortem examination showed a purulent meningitis over the cerebral hemisphere and base of the brain, as well as recent hemorrhages over the right temporal and parietal lobes, the pons, and the adjacent cerebellar portions. Histologic examination showed the residues of extensive destruction of the labyrinth. Nothing was preserved of the membranous labyrinth. All the cavities were filled with newly formed connective tissue and bone, which far exceeded the boundaries of the old labyrinth cavities, so that a large portion of the old capsule bone was comprised in them. The condition existed which has been designated by Zange as labyrinthopathy, and by Wittmaack as labyrinth phthisis.

SEROUS OR PROTECTIVE MENINGITIS [107]

Protective meningitis [108] means a protective or walling-off inflammation of a small portion of the dura outside of the subarchnoid spaces; it occurs in sinus thrombosis, brain abscess and Gradenigo's syndrome.

Ferreri reported a case of a circumscribed collection of cerebrospinal fluid on the convexity of the cerebellum, in a patient who had suffered for a long time from Ménière's disease.[109]

The functional examination showed a lesion of the right half of the cerebellum, on the side of the diseased ear, with hyporeflexia of the vestibulocerebellar tracts associated with integrity of the vestibulo-ocular tracts. Retropyramidal trephining, with incision of the cerebellar dura and separation from the bone, resulted in recovery.

Ferreri thinks that the vertiginous manifestations were due to hyperpressure on the vestibulocerebellar tracts.

Serous Meningitis from Labyrinth with Aphasia.—Brémond and Pourtal [110] reported the following case:

A girl, aged 16 years, after an operation with jugular ligature in sinus thrombosis suffered periodically for three years from suppuration of the right ear. On admission, in addition to an acute exacerbation, she had right-sided peripheral facial paralysis, complete aphasia with word deafness and word blindness, and agraphia. Severe vertigo and vomiting developed. She had a slight headache, but no paralytic phenomena. The labyrinth was opened. Lumbar puncture yielded an opalescent fluid containing albumin and polymorphonuclear cells. No organisms were present. There was a slow decrease in temperature with disappearance of the facial paresis and subsidence of the aphasia. The patient recovered.

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107. Eagleton, Wells P.: Brain Abscess, New York, The Macmillan Company, 1922, p. 150.

108. Wright, C. D'A.: Protective Meningitis, Journal-Lancet **5**:499 (Oct. 15) 1930.

109. Ferreri, G.: Operated Case of Otogenic Serous Meningitis of the Posterior Cranial Fossa, by the Retro-Pyramidal Route, Riv. otol. **6**:239, 1929.

110. Brémond, Roger and Pourtal: Aseptic Suppurative Meningitis of Right Sided Labyrinthine Origin, with Aphasia, Rev. d'oto-neuro-opht. **7**:41 (Jan.) 1929.

Slight, or absence of, headache, in spite of grave meningitic symptoms, is of favorable prognostic significance.

The appearance of aphasia with right-sided labyrinthitis in a right-handed person is rare. Isolated meningitic foci, or a serous meningitis of the left temporal convolutions, may furnish the explanation.

Circumscribed Meningitis Cured by Intracranial Injections for Diagnostic Purposes When Brain Tumor Was Suspected.—Moniz[111] used carotid injections on both sides at an interval of eight days. Up to 1929, he has given eighty injections to forty patients, without any outward complication. It may happen that the injection is followed by a rapid and considerable decrease of the headaches, and by an improvement in the vision. This was true in five cases which perhaps were cases of circumscribed serous meningitis.

Meningism.—Layton[112] gives Dupre's definition of meningism: "The syndrome of symptoms aroused by the irritation of the meningocortical regions and independent of any anatomico-pathological change." He contended that the so-called "meningism" is the earliest stage of meningitis. The only symptom of any importance is rigidity of the neck. By the time there are abdominal and other reflexes, the chances of recovery are very small. Therefore, whenever there is rigidity of the neck, in a case of otitis, the condition should be regarded as meningitis. Layton thinks that lumbar puncture should not be done because by it organisms are sucked from the neighborhood of the ear to the region of the foramen magnum, and the infection distributed.

Bulbar Cisternal Meningitis.—Two cases of caries of the petrous apex from suppurative otitis, reported by me,[113] present the suggestive "pain behind the eye," associated with a low grade chronic sepsis and recurrent attacks of slightly stiff neck.

In one case the prodromal signs lasted sixteen days, and in the other, forty-three days before the onset of symptoms of bulbar cisternal meningitis.

The fluid from the lumbar region was cloudy, but contained no organisms. Autopsy in both cases disclosed a large carious cavity involving the petrous apex and extending onto the anterior and posterior surfaces of the petrous pyramid, causing a localized meningitis of the pontile cisterna followed as a terminal process by a general suppurative meningitis.

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11. Egas, Moniz: Arterial Encephalography as a Method for the Localization of Brain Tumors, Klin. Wchnschr. **8**:1118 (June 11) 1929.

112. Layton, T. B.: Otitic Meningism, J. Laryng. & Otol. **44**:246 (April) 1929.

113. Eagleton, Wells, P.: Localized Bulbar Cisternal (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:386 (March) 1930.

Two additional cases of bulbar meningitis of traumatic origin are also reported, a clinical diagnosis of bulbar meningitis having been made during life because both patients presented similar clinical pictures.

Autopsies confirmed the diagnosis that the meningeal process in the early stage was confined to the bulbar cisterna.

The symptoms of localized bulbar meningitis of otitic origin can be summarized as: (1) a period with signs of dural irritation of the middle fossa, of which facial pain, especially pain behind the eye, is the most significant (possibly associated with abductor paralysis), followed by (2) symptoms of arachnoid inflammation of the posterior fossa near the median line. Localizing middle and posterior fossa symptoms are thus (3) in combination, one with the other. In addition there is a state of (4) semicoma from which the patient can easily be aroused, associated with (5) the supine position of the patient (on his back), with eyes closed and (6) intermittent recurrence of vertical nystagmus. All these are signs of posterior fossa involvement, but are not accompanied by the severe headache, great restlessness, or the opisthotonos so usual with cortical meningitis. (7) Repeated trials of visual fields may reveal great bilateral variations and the sudden appearance of a bitemporal hemianopia somewhat resembling pressure from a pituitary tumor.

Latent Meningitis from the Ear in Children.—Dabney [114] has seen six cases of otitis media in children ending in death from meningitis. None of the usual signs of meningitis was present, though headache occurred in all. Moreover, no sign appeared sooner than ten days after operation. In fact, three of them showed no localizing signs until four weeks later, unless the existing headache can be so regarded.

Absence of signs of meningitis in these cases before operation is probably due to localization of the process. After operation it is probably due to the decompression effect of mastoidectomy.

Proteus Meningitis from the Ear.—Kortenhaus [115] described a case of otogenic meningitis by an indol-negative Proteus strain, the sixth human case of Proteus meningitis on record since the discovery of this bacterium by Hauser in 1885. The postmortem diagnosis was chronic middle ear suppuration, meningitis and brain abscess (temporal lobe), both following a radical mastoid operation.

Unrecognized Sinusitis as the Cause of Meningitis.—Autopsy revealed,[116] besides the purulent leptomeningitis, a collection of pus in

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114. Dabney, V.: Latent Meningitis Complicating Surgical Mastoiditis; the Significance of Headache; Limitation of the Pediatrician's Attendance, Ann. Otol., Rhin. & Laryng. **39**:527 (June) 1930.

115. Kortenhaus, D.: Proteus Meningitis by Way of the Ear, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **19**:146 (Jan.) 1930.

116. Geschelin, A. I.: Purulent Meningitis of Unknown Origin, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **126**:230 (Oct.) 1930.

the right frontal sinus. The pus was sterile and the walls of the sinus were intact.

Fulminating Meningitis Following Ethmoidectomy.[117]—After the removal of the right middle turbinate the anterior and posterior ethmoid bones were seen to be diseased. The ethmoids were evacuated. Purulent meningitis supervened some hours later. The patient died at the end of three days.

Three cases with death from meningitis after accessory sinus empyema, all taking a fulminating course, are reported by von Eicken.[118]

Case 1.—The patient was admitted with swelling about the eye. He had a history of chills and high temperature. An incision was made, as the condition was assumed to be a furuncle. The roentgen picture showed darkening of the maxillary antrum and of the frontal sinus on the left side. The maxillary antrum was opened. Another opening was made in the floor of the frontal sinus, after removal of the anterior ethmoid cells. A few days later repeated chills occurred; the child vomited. Meningitis was confirmed by lumbar puncture.

Removal of the posterior wall of the frontal sinus liberated pus under high pressure. Autopsy showed that the extension into the meninges had occurred by way of an orbital recess.

Case 2.—The patient presented the symptoms of maxillary sinus suppuration and suppuration of the ethmoid cells. The maxillary antrum was opened. The ethmoid cells were exposed, without removal of the mucosa. The patient's condition improved and all danger was believed to be over. A few days later, there was a rise of temperature followed by fulminating meningitis. Death occurred in two days.

Case 3.—The patient was admitted to the hospital with a high temperature; he had suffered from chills, but no vomiting. There was edema over the right upper eyelid. When the right upper maxillary antrum was opened, the mucosa at the orbital wall was found to be swollen. No pus was found in the maxillary antrum. The frontal sinus was explored, as were the ethmoids. The next day the patient had slight rigidity of the neck, no Kernig sign but severe headache. On lumbar puncture, the cerebrospinal fluid contained blood. The pressure was not increased. The blood agar plates were sterile. The patient was slightly confused. A rise of temperature occurred on the second day and the patient rapidly failed.

Meningitis Secondary to Frontal Sinusitis with Latent Osteomyelitis.—Ballenger[119] reported a case of double frontal sinusitis with suppurative meningitis (Staphylococcus aureus), complicated by bronchopneumonia and multiple abscesses of the scalp, probably of osteo-

117. Wilkerson, W. W., Jr.: Meningitis Following Sinusitis, Laryngoscope **40**:705 (Sept.) 1930.

118. von Eicken: Meningitis in Frontal Sinusitis, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **18**:340, 1929.

119. Ballenger, Howard C.: Meningitis (Staphylococcus Aureus) Secondary to Sinusitis with Operation and Recovery, Arch. Otolaryng. **11**:385 (March) 1930; Meningitis (Staphylococcus Aureus) Secondary to Frontal Sinusitis, J. A. M. A. **94**:1040 (April 5) 1930.

myelitic origin, with septicemia and drainage of pus through the posterior wall of the frontal sinus, followed by recovery.

The patient, a boy, entered the hospital with convulsions and swelling of the forehead and left eyelid. The convulsions lasted about two hours. The illness began two days before admission, with a frontal headache, fever, and a purulent discharge from the nose.

Examination revealed a diffuse, brawny, tender swelling, without redness or fluctuation, over the lower portion of the forehead and the upper part of the nose and left upper eyelid. Rigidity of the neck was present; the knee reflexes were not elicited; Kernig's sign was positive.

The spinal fluid was turbid and under moderate pressure. The cell count was 320, with 96 per cent polymorphonuclears and 4 per cent lymphocytes. Culture of the spinal fluid showed gram-positive staphylococci, which proved to be Staphylococcus aureus.

At operation, the left frontal sinus was found filled with yellow pus. The mucous membrane appeared to be partly destroyed. After the inner table of the frontal sinus had been removed, thick yellow pus escaped from within the dura. Culture revealed Staphylococcus aureus. Pus was also found in the right frontal sinus.

Cultures from the frontal sinus, dura, blood and nose all showed Staphylococcus aureus.

Three days after the operation, pneumonia developed, which was probably of a metastatic or a hypostatic type.

The highest white blood cell count was 32,400.

A week later two abscesses developed over the occipital region and the parietofrontal regions. Roughened bone could be felt under each abscess. Positive blood cultures of Staphylococcus aureus were obtained twice.

The patient was in a semicomatose state, drowsy and irrational at times during most of his illness. There were occasional attacks of vomiting. Many times he would waken with a loud cry (meningeal cry). On the twenty-first postoperative day, there developed complete paralysis of all the muscles of the right eye with a dilated pupil, which did not react to light.

Recurrent Attacks During Seven Years of Meningitis Originating from the Nose.—In the case reported by Bronstein [120] the first three attacks of meningitis occurred within a year, and the fourth after a lapse of seven years.

The patient was admitted to the hospital in September, 1920. He had had a nose and throat operation nine days before admission. Following this a discharge appeared from the right nostril. There were a positive Kernig sign and negative Babinski and Brudzinski reflexes. Some rigidity of the neck was present. Spinal puncture revealed a cloudy fluid under increased pressure. The cell count was 20,000, chiefly polymorphonuclear leukocytes. No organisms were found. Repeated spinal punctures were done, and antimeningococcus serum was given intraspinously. The patient was discharged in five weeks, with a final diagnosis of suppurative cerebrospinal meningitis of unknown etiology.

He was readmitted to the hospital on Feb. 4, 1921. A few days prior to the second admission a nasal discharge appeared and then ceased, followed by severe

120. Bronstein, I. P.: Recurrent Meningitis Within a Period of Eight Years: Report of an Unusual Case, J. A. M. A. **92**:2094 (June 22) 1929.

headaches. Projectile vomiting occurred, without previous nausea. The pupils were contracted, but they reacted slowly to light. The Babinski and Kernig reflexes were negative; the knee-jerks, markedly increased. Spinal puncture revealed turbid fluid under increased pressure. Antimeningococcus serum was given. Several days after admission, definite rigidity of the neck and back, with opisthotonos developed. The ethmoid labyrinth on the right side was partially removed, and the anterior wall of the right sphenoid sinus was resected. Direct smear of the spinal fluid gave gram-negative intracellular diplococci and a few extracellular cocci. Staphylococci and streptococci were present, but no meningococci. The white blood count on admission was 29,300. Repeated spinal punctures were made and antimeningococcus serum given intraspinally. The patient was discharged on February 18, as improved, with a diagnosis of epidemic (?) cerebrospinal meningitis.

The patient was admitted again on Aug. 1, 1921, after being well until a few days before. There was a nasal discharge for a week before admission, which ceased before he entered the hospital. He suffered from severe headache and projectile vomiting. The Kernig and Babinski reflexes were negative. The spinal fluid was cloudy, under increased pressure and contained no organisms. The day after admission rigidity of the neck appeared, which was relieved by spinal puncture. Antimeningococcus serum was given. Smears of the nose and throat showed hemolytic streptococci. Several punctures were made and serum given. The patient was discharged on September 11, as improved, with diagnosis of recurrent meningitis.

He was admitted for the fourth time on Dec. 16, 1928, with a diagnosis of meningitis. The Brudzinski reflex was positive; the Kernig and Babinski reflexes on the left were positive. The patient was in a semistuporous state; he had severe headache. The attack was preceded by mild influenza lasting three days. The neck was rigid; the pupils were small and equal and reacted sluggishly to light; the back was slightly arched. The sinuses, ears and nasal cavities were normal. Spinal puncture gave a cloudy fluid under increased pressure, with 814 cells and a positive globulin reaction. Smear of the spinal fluid showed chiefly polymorphonuclear leukocytes and a moderate number of intracellular diplococci. Antimeningococcus serum was given. Bacteriologic examination revealed hemolytic streptococci in pure culture. A typical meningitic curve was obtained in the spinal fluid. The patient died.

In this case the original attack was associated with a nasal operation. The association with a nasal discharge was noted in the second and third attacks, whereas in the fourth attack the nasal discharge was absent but influenza was present. The causative organisms varied, but at no time was the identity of meningococci definitely established.

From reading the case, I am of the opinion that the patient had a low grade osteomyelitis of the sphenoidal basis which was repeatedly brought into activity by recurrent infections.

Meningitis Through an Old Fracture of the Labyrinth.[121]—A child suffered from meningitis originating in an acute recurrence of a middle

121. Meyer, M.: Unusual Origin of Otogenic Meningitis Through Traumatism with Delayed Onset of "Croupous" Meningitis, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **30**:375, 1929.

ear inflammation. There was a history of traumatism four weeks prior to the acute otitis media from a fall on the head, which showed no clinical symptoms after death. The histologic examination revealed several still unhealed fissures in the medial tympanic wall, which were only united by connective tissue. Through these, an acute otitis had invaded the labyrinth.

Ununited Fracture of the Labyrinth the Cause of Meningitis.—Nager [122] reported the following case of delayed meningitis after fracture of the labyrinth:

> A man sustained a fracture of the base of the skull followed by unconsciousness. There was complete deafness on the right side, with loss of vestibular function. A roentgenogram showed a transverse fracture of the petrous bone. During the eighth week symptoms of meningitis appeared, which cleared up in six weeks after repeated lumbar punctures. Two months later, the patient again had meningitis. He died three days later, i. e., 190 days after the injury.
>
> Autopsy showed meningitis with an apparently healed fracture of the right temporal and left occipital bones. That part of the fracture lying in the endochondral bone showed only fibrous union. There was a fistula between the middle ear and the cochlea, with a cyst at its cochlear end. It was evidently from this point that the infection had reached the inner ear. The labyrinth contained purulent exudate which had passed from here through the internal auditory meatus to the meninges.

Prognosis and Results of the Surgical Treatment of Meningitis.—Statistics in the forty cases of otogenic meningitis from the Rigshospital in Copenhagen, Denmark, from 1910-1927,[123] show that of nineteen cases of tympanogenic meningitis, ten were fatal, and of twenty-one labyrinthogenic cases, twelve were fatal. There were twice as many males as females. The mortality for men was 59.2 per cent; for women 46 per cent. Meningitis is most common between the ages of 5 and 25 years. The prognosis is worse between 5 and 15, and 40 and 60 years. The prognosis is bad (1) in the presence of positive bacterial findings in the spinal fluid, (2) in cases with white cell counts above 5,000, (3) in cases with polymorphonuclear leukocytes rather than with monocytosis in the cerebrospinal fluid, and (4) in acute middle ear suppuration rather than in chronic middle ear suppuration. Labyrinthogenic meningitis originating as a sequel to acute middle ear suppuration has the gravest prognosis; death followed in six of seven cases.

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122. Nager, F. R.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:217 (Aug.) 1929.

123. Schroeder, R.: Results of Treatment of "Pure" Otogenic Meningitis in the Oto-Laryngological Department of the Copenhagen Rigshospital in 1910-1927, Hospitalstid. **72**:605, 1929.

Prevention of Meningitis in Accidental Operative Injuries to the Dura.—As shown by Schlander,[124] small accidental tears in the dura are more dangerous than large ones. In the course of operations, he transforms small unintentional dural lacerations into larger ones by widening the hole. The lesser danger of the larger dural lesions (already emphasized by Zange in traumatic cranial injuries) is due to the fact that the larger gaps can be more rapidly closed off by apposition of the meninges to the dura. Smaller dural lesions, in the author's opinion, are dangerous because occlusion fails to occur, and on the contrary, the cerebral fluid is aspirated, as it were, by pulsations of the brain, and thus infection travels from the outside into the interior of the cranium.

TREATMENT FOR MENINGITIS

Influence of Ventricular and Cisternal Puncture on the Direction of Flow in the Treatment for Meningitis.—Sachs, Wilkins and Sams [125] summarized their study on the cerebrospinal circulation as follows:

> The reduction of pressure by lumbar puncture creates an artificial circulation toward the point of puncture. If one desires to inject serum with the idea of having it reach all parts of the central nervous system, it is much more effective to inject it into the ventricles or basal cisterna. In meningitis, repeated spinal punctures or permanent lumbar drainage tends to spread the infection by producing an artificial circulation. If drainage is to be employed, it is more effective to apply it in the region of the basal cisterna than in the lumbar region.

Rendering the Blood Hypotonic in the Treatment for Meningitis.—In 1926, Kubie described a method for drainage of the central nervous system, which he believed might be effective in certain infections of the nervous system.[126]

The theory is as follows:

> The cerebrospinal fluid is a dialysate derived from the blood plasma and is in hydrostatic and osmotic equilibrium with it. The cerebrospinal fluid is formed by a process of filtration through the capillaries of the choroid plexus and also through the capillaries throughout the central nervous system, the latter portion of the fluid draining into the subarachnoid space by way of the perivascular spaces. Drainage of the cerebrospinal fluid by lumbar puncture, therefore, should drain the perivascular spaces, as well as the subarachnoid space and ventricles. The fluid formed from the choroid plexuses and the perivascular fluid may be increased either by raising the hydrostatic pressure in the capillaries or by rendering the blood hypotonic. From a theoretical consideration of these two methods, the latter should be by far the more effective.

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124. Schlander, E.: Operative Injury of the Dura, Monatschr. f. Ohrenh. **64:** 757 (July) 1930.

125. Sachs, E.; Wilkins, H., and Sams, C. F.: Studies on Cerebrospinal Circulation by a New Method, Arch. Neurol. & Psychiat. **23:**130 (Jan.) 1930.

126. Fremont-Smith, Frank; Putman, Tracy J., and Cobb, Stanley: Forced Drainage of the Central Nervous System: Its Effect on the Blood and on the Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **23:**219 (Feb.) 1930.

Methenamine in Experimentally Produced Meningitis.—In Starlinger's [127] experiments, a combination of a 40 per cent solution of methenamine administered suboccipitally, and a 10 per cent solution administered intravenously, associated with cisternal puncture, was effective in curing one of the four dogs in which a streptococcic meningitis was proved to exist. In two cases sterile cerebrospinal fluid was noted at necropsy.

Surgery of the Labyrinth in the Treatment of Meningitis.—Kopetzky and Almour [128] discussed the possibility of the spread of a purulency into the subarachnoid space through the internal auditory meatus, the aquaeductus cochleae or the ductus endolymphaticus. In a purulent labyrinthitis there are increased cells in the fluid, increased pressure, cloudiness, traces of albumin, and sometimes globulin, a normal sugar content and no bacteria. Such a fluid, found in the presence of a clinically diffuse labyrinthitis, indicates suppuration within the labyrinth. A normal spinal fluid, with but a slight increase in pressure, indicates labyrinthitis of a serous nature. Extension of the circumscribed purulent labyrinthitis beyond the protective zone of granulations is indicated by a change in the nature of the fluid.

When there exists no threat to the meninges, surgical intervention on the labyrinth is not required. When a meningitis exists as the causative factor of the diffuse purulent labyrinthitis—when the lesion in the inner ear is secondary to a purulent leptomeningitis—surgical intervention on the labyrinth per se is not indicated. When a circumscribed purulent labyrinthitis exists, removal of the causative factor is sufficient to stop the progress of the lesion in the capsule. In diffuse serous labyrinthitis, removal of the causative factor in the mastoid and middle ear will result in rapid cessation of the disease.

If, in connection with diffuse labyrinthitis, there exists a definite reaction in the meninges, it is essential to drain the cisterns. In the event of a purulent diffuse labyrinthitis with threatened meningeal involvement, surgical intervention on the labyrinth may cause a more rapid spread of the infection into the subarachnoid space by the traumatic disturbance of the limiting protective forces. It is better to drain the meninges early than to await the appearance of a suppurative meningitis.

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127. Starlinger, F.: Experimental Treatment of Suppurative (Purulent Meningitis), Arch. f. klin. Chir. **156**:474 (Dec. 10) 1929; abstr., J. A. M. A. **94**:1103 (April 5) 1930.

128. Kopetzky, S. J., and Almour, R.: Surgical Indications in Purulent Labyrinthitis and Otitic Meningitis, Laryngoscope **39**:228 (April) 1929.

Meningitis from Apex Suppuration.—Of the four patients with petrous apex suppuration, whose cases were reported by me,[129] one had meningitis with bacteria in the lumbar fluid at the time of operation; the patient died. In another case, the operation precipitated a general meningitis, with streptococci in the fluid; the patient recovered, however.

Operative Procedure in the Surgical Treatment of Meningitis.—The posterolateral two thirds of the petrous pyramid is unlocked. (1) The posterior buttress is entirely removed, (2) the angle of the petrosal lowered, and (3) the bone covering the sinus and that over the cerebellum are sacrificed, as well as the bone of the squama and the tympanic ring.

The extensive bony destruction allows (4) displacement backward of the lateral sinus and (5) elevation of the superior petrosal upward. This permits an investigation (6) of the dura of the posterior fossa as far as the region of the saccus endolymphaticus and (7) of the floor of the middle fossa inward to well above the labyrinth. The depth of separation of the dura from the bone is dictated by the degree of intradural pressure.

Following failure to find an area of bony pathology (*a*) over the cerebellum or (*b*) of the middle fossa with (*c*) change in the dura or (*d*) evidence of thrombosed veins leading from the bone into the dura, (8) the dura in the region of the saccus endolymphaticus is nicked. A large proportion of cases show free pus or purulent fluid in this area in the early stages of meningitis.

(9) The arachnoid prolongation surrounding the internal auditory meatus is now opened.

The "nicking" of the dura is watchmaker's work, made possible by the removal of the large area of bone which permits of sufficient displacement of the brain.

The evacuation of fluid should be done entirely by sight and should be bloodless. The intradural veins are easily torn; hemorrhage then obscures the field. Intra-arachnoid bleeding has a most deleterious influence and has been the deciding factor against recovery, as shown by many postmortem examinations.

Hemorrhage into the arachnoid space of the posterior fossa has a tendency to pass above the superior surface of the cerebellum and through the tentorial opening above the fourth ventricle.

In cases of great tension, the internal auditory meatus has to be opened by a "harker" with its point toward the posterior surface of the petrous pyramid.

If the infection comes from a suppurating labyrinth, (*a*) the vestibule is opened posteriorly (Neumann), followed by (*b*) destruction of the vestibule and the cochlea, and (*c*) drainage through the modiolus (West and Scott).

A collection of fluid within the dura of the middle fossa rarely occurs, excepting when the infection follows a thrombosed vein above the labyrinth, or is the result of caries of the superior semicircular canal, when there may be a collection of fluid in the subdural space.

(10) In the cases of meningitis from caries of the petrous apex, the operation of the unlocking of the anterior portion of the petrous pyramid is completed. This allows (*a*) the plugging of the canal of the middle meninges or the "clipping" of the artery, (*b*) after which elevation of the dura permits inspection of the apex.

(11) The suppurative lesion in the petrous apex is drained, and (12) the dura is opened just below the gasserian ganglion, thus evacuating the fluid from the prolongation around the roots of the fifth nerve which extends forward from the posterior fossa.

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129. Eagleton, Wells P.: Unlocking the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex: Report of Four Cases with Recovery in Three, Arch. Otolaryng. **13**:386 (March) 1931.

In some cases another opening has been made below the superior petrosal sinus. In exploration of the frontal region, (1) all the bone of the forehead is removed, and (2) the dura is elevated over the orbit. (3) A small nick is made in the subdural space behind the frontal sinus, or the chiasmal cistern is evacuated.

Subarachnoid Irrigation with Ringer's Solution.[130]—Irrigation with Ringer's solution was used in three cases of meningitis. Cure was obtained in two cases in which the irrigation was made through a suboccipital puncture; the third case (irrigation by the lumbar route) terminated in death. Autopsy showed grave internal hydrocephalus, and adhesions in the neighborhood of the foramen magnum. Fluid obtained by suboccipital puncture is more changed in purulent meningitis than the fluid from lumbar puncture. In the healing stage of purulent meningitis, a thick coagulate often appears from two to four hours after withdrawal of the purulent fluid.

Subarachnoid Irrigation and the Injection of Iodoform.—Jenkins [131] discussed the following: (1) a method of washing out the subarachnoid space in cases of septic meningitis secondary to infection of the ear; (2) the value of maintaining a positive pressure of the cerebrospinal fluid when operating on a septic region communicating with the subarachnoid space and (3) the operative treatment when cerebrospinal fluid is escaping from the ear.

In most cases, in addition to simple drainage of the subarachnoid space, Jenkins washes out the space and introduces a small quantity of specially prepared iodoform which has been washed free from the perchloride with modified Locke's solution.

The technic is as follows:

Superior and inferior labyrinthotomy are performed. The wound is syringed with 2 per cent alcoholic solution of iodine. A fine needle is passed into the labyrinth, and the interior is well douched with the iodine solution. The internal auditory meatus is now opened. A lumbar puncture is performed.

When a flow of cerebrospinal fluid is established, the lumbar puncture needle is connected with a reservoir of the modified Locke's solution. A strand of silkworm-gut is put into the internal auditory meatus to help the flow of cerebrospinal fluid.

After a few ounces of Locke's solution has passed, the serum, carrying about 8 grains of specially prepared iodoform, is injected through the lumbar puncture needle, and the Locke's solution is again allowed to flow until fluid is returned clear at the ear.

If the internal auditory meatus is blocked by swelling, the cisterna pontis may be reached in front of the cerebellum. The lateral recess of the cisterna pontis extends a little beyond the opening of the internal auditory meatus, and is tapped. The wound is then lightly packed with specially prepared iodoform gauze. In some patients subarachnoid irrigation has to be done two or three times.

130. Mann, W.: Treatment of Purulent Meningitis, Deutsche med. Wchnschr. **56**:1306 (Aug. 1) 1930.

131. Jenkins, G. J.: Some Points in the Treatment of Meningitis, J. Laryng. & Otol. **44**:230 (April) 1929.

Meningitis Treatment by Chemotherapy and Immunotherapy.—Mauthner [132] pointed out that a sudden and rapidly progressive infection of the meninges may originate not only through the internal ear but also by continuity of suppuration, especially as a hematogenic infection. He asked for the employment of chemotherapeutic and immunotherapeutic measures in the following cases: (1) in the presence of a preceding traumatism, (2) after paracentesis when the fever persists or assumes a septic or pyemic character and (3) in all streptococcus infections.

Intravenous Injections of Sodium Chloride Solution.—Stewart [133] based his study on 114 cases of acute purulent meningitis following middle ear disease. He thinks that operative treatment should be combined with chemotherapy. Fifty cubic centimeters of 10 per cent sodium chloride solution is given intravenously daily. In one case, irrigations from the lateral ventricles to the lumbar region gave disappointing results.

Intravenous Injections of Methenamine.—Rimini [134] discussed the therapeutic value of lumbar puncture, which was performed ten times in his case, as well as the danger of a sudden lowering of the pressure. Among medicinal substances, methenamine is primarily capable of penetrating through the filter of the choroid plexus. In the author's case, 10 cc. of a 40 per cent solution of methenamine were repeatedly injected intravenously.

Methenamine by Intravenous and Intralumbar Injections.—Rejtö [135] reported the following case in which methenamine was used:

> The patient suffered since childhood from bilateral ear suppuration. Operation was performed on account of acute exacerbation, unconsciousness, vomiting and chills. Six days after the operation a temporal lobe abscess developed, which was drained; four days later, meningitis developed. Lumbar puncture revealed streptococci. After eight days the patient's condition improved. Pus continued to be voided from the abscess for two weeks. The patient recovered, following an attack of endocarditis.

Methenamine was utilized in the treatment, 5 cc. in intralumbar injection every other day, and 10 cc. intravenously on the intervening days.

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132. Mauthner, O.: Clinical Studies on Otogenic Meningitis, Monatschr. f. Ohrenh. **64**:1016 (Sept.) 1930.

133. Stewart, J. P.: Acute Purulent Meningitis of Aural Origin, J. Laryng. & Otol. **44**:225 (April) 1929.

134. Rimini, E.: Labyrinthogenic Meningitis: A Case Operated Upon and Cured, Arch. internat. de laryng. **8**:262 (March) 1929.

135. Rejtö, A.: Cured Case of Temporal Lobe Abscess and Meningitis, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **15**:217, 1930.

136. Footnote 136 has been deleted by the author.

Intravenous Injections of Antipneumococcic Serum.—In a case reported by Laignel-Lavastine and Bernal, the treatment consisted in the administration of antipneumococcic serum.[137] At the time of antrotomy, two days later, after the cerebrospinal fluid had cleared, no pathologic change was found in the mastoid process or the antral cells. On the fifth day, the temperature was normal.

Intra-Arachnoid and Intravascular Treatment for Meningitis.—Eagleton, Dearmin and Peers' experiences with injection of serum and antiseptics into the arachnoid cavity [138] or into the carotid [139] have failed to produce any beneficial effects, but several disastrous effects have resulted.

My belief is that in the future intradural treatment will be by way of the injection of a bacterium or a bacteriophage—not a chemical irritant—into the cerebrospinal system. This may be beneficial, as I have seen marked modification of the activity of one type of bacteria by attenuation and thus loss of virulence, by the presence of another micro-organism in the arachnoid mesh.[140]

Disastrous Results Following Intracarotid and Intracisternal Medication.—Dearmin [141] treated two patients who had *pneumococcic meningitis, by intracarotid and intracisternal* injections of pneumococcic antibody solution and 1 per cent optochin hydrochloride.

Case 1.—On Oct. 3, 1929, the patient, aged 20, complained of pain in the right ear and of headache. On October 9, he had a severe headache and became delirious. He had one convulsion while being taken to the hospital. On admission he was in a deep coma. The right ear showed a sagging of the posterior canal wall and a slight amount of thick exudate covering the drum. The neck was rigid. There was a bilateral positive Kernig sign. The deep knee reflexes were hyperactive. The pupils were dilated but reacted to light. The right disk was pale. The white blood count was 12,250 with 90 per cent polymorphonuclears. By lumbar puncture about 7 cc. of very cloudy spinal fluid were withdrawn. A smear showed pneumococci.

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137. Laignel-Lavastine and Bernal: A Cured Case of Otitic Purulent Pneumococcus Meningitis, Bull. et mém. Soc. méd. d. hôp. de Paris **53**:1283 (Nov. 25) 1929.

138. Eagleton, W. P.: Cerebrospinal Fluid as an Aid to Diagnosis and Treatment of Inflammatory Diseases: Injection of Bile, J. Laryng. & Otol. **44**:732 (Nov.) 1929.

139. Dearmin, R. M.: Meningitis of Otitic and Nasal Origin, Thesis submitted to the Faculty of the Otolaryngologic Section of the Graduate School of Medicine, University of Pennsylvania, in partial fulfilment of the requirements for the degree of Master of Science for two years of graduate work in Otolaryngology, 1928-1930, p. 732.

140. Malis, S.: Pneumobacillus (Friedländer's) Meningitis Secondary to Otitis Media, Arch. Otol. **12**:162 (Aug.) 1930; case 2.

141. Dearmin.[139]

An extensive mastoid operation was done. The superior petrosal angle of the petrous pyramid was removed, and the lateral sinus exposed from the jugular bulb almost to the torcular Herophili. The sinus wall was gangrenous, as was also the dura over the anterior surface of the cerebellum. A large clot was removed from the lateral sinus. The dura was opened over the anterior surface of the cerebellum and over the middle fossa, from which a localized collection of cloudy fluid was drained.

On October 10, on lumbar puncture, only 2 cc. of bloody spinal fluid was obtained.

On October 11, by cisternal puncture, 20 cc. of cloudy fluid was withdrawn; 20 cc. of the antibody (Huntoon's pneumococcus) solution with 1 per cent optochin hydrochloride was injected into each carotid artery, after which 10 cc. of the same solution was allowed to flow into the cisterna magna. About five minutes later the breathing became shallow, and fifteen minutes later ceased. The heart continued to beat for about two minutes.

Case 2.—The patient, aged 3, two days after being in swimming, had severe pain in the left ear. The ear discharged for about one week. Later occipital headache developed. A roentgenogram showed congestion of the left mastoid. Myringotomy was done but there was no discharge.

On Oct. 10, 1929, sudden excruciating pain developed in the left side of the head and a throbbing pain deep behind the left eye. The child had a chill and high fever.

On October 12, on admission to the Newark Eye and Ear Infirmary, the drum bulged in the posterior half. A myringotomy was followed by bleeding but no pus. The white cell count was 11,600 with 79 per cent polymorphonuclears and 21 per cent lymphocytes. Neurologic examination gave negative results.

On October 14, a slight chill developed; the temperature was 101 F. A radical mastoidectomy was done and the entire angle of the petrous pyramid removed. The sinus was found to be thrombosed from the torcular Herophili to the jugular bulb. The jugular was tied. Blood culture showed a type III pneumococcus. After operation, the patient was drowsy but complained of no pain. The temperature remained around 101 F. during the entire course of the disease.

On October 17, the patient had a stiff neck and a Kernig sign. The veins of both fundi were engorged and both nerve heads indistinct. Lumbar puncture showed a turbid fluid under increased pressure. There was no reduction of Fehling's solution. Examination of the fluid showed: albumin, plus; globulin, plus; and a cell count of 8,200. Smear and culture showed a pneumococcus, type III.

Intracarotid injections were made. The common carotid artery was exposed. A cisternal puncture was done, and about 20 cc. of fluid was withdrawn. Fifteen centimeters of pneumococcic antibody solution (Mulford) with 1 per cent ethylhydrocupreine hydrochloride was injected into the carotid. The other carotid was not exposed; the same amount was injected by the technic advised by Hirsch, Myerson and Halloran.[142] Five centimeters of serum was injected into the cisterna magna. Within ten minutes the patient had a convulsion, which started in the facial muscles and extended downward to the lower extremities. Breathing became shallow. In ten minutes he had another convulsion and breathing ceased, but was restored by means of artificial respiration and stimulation with carbon dioxide.

142. Hirsch, H. L.; Myerson, A., and Halloran, R. D.: Boston M. & S. J. **192**:712 (April 9) 1925.

Within an hour he had five convulsions; each time the respirations ceased. After the last convulsion the respirations could not be restored. The pulse could be felt for about two minutes.

The case of a patient treated by intraspinal and intravenous injection of antipneumococcus serum was also reported by Dearmin.

The patient, aged 6 years, complained of a headache and pain above the left eye. He had had a slight head cold previously. He became delirious and had a high fever. The following day he vomited and was stuporous and restless The next day the temperature was 105 F., respirations 52 and the pulse was rapid and thready. At times he was rational. He was given an immediate hypodermoclysis of 600 cc. of 10 per cent dextrose. At 7 p. m. he had definite signs of meningitis, i. e., a positive Kernig sign, opisthotonos, strabismus and dermographia. The spinal fluid was cloudy but not under pressure. It contained 680 cells; globulin, 2 plus, and a gram-positive diplococcus with a capsule. A majority of the cells were lymphocytes. On culture, pneumococcus type II was found. The patient was given 10,000 units of Felton's antipneumococcus serum intraspinally, and 10,000 units intravenously, as well as 0.5 Gm. of quinine by rectum.

Immediately following, the patient had a severe chill, the body became cold and clammy, and he became pulseless. He remained in this condition for about eight hours. At 4 p. m. the following day he had Cheyne-Stokes breathing, and at 5 p. m. the respirations ceased, although the heart continued to beat for a short time.

Autopsy showed that the cortex of the brain was covered with a yellowish-green exudate, confined to the pia-arachnoid, which extended over the frontal and temporal lobes on the left side and over the tip of the frontal lobe on the right side. The base of the brain showed an exudate on the inferior surface of the left frontal lobe, which was not confined to the pia-arachnoid but extended to the dura in this location. The entire cortex showed a beginning encephalitis. The sphenoid sinus on the left side as well as the ethmoids were filled with polyps, which were riddled with small pockets of pus similar to that seen in the brain. The mucous membrane of the sphenoids and ethmoids was thickened and edematous. The right sinuses were also filled with polyps, but no pus was seen.

Comment: Death in the foregoing three cases was apparently due to respiratory paralysis, although in the last it was the type of death that might be expected from an acute meningitis. The respiratory failure may have been due to a specific action of the serum and ethylhydrocupreine hydrochloride on the tissue of the central nervous system. Extremely small doses of the solution should be used in the cisterna magna until tolerance is established. Goldberg [143] reported one case of pneumococcic meningitis in which he injected 10 cc. of 5 per cent of ethylhydrocupreine hydrochloride intraspinally after withdrawing 10 cc. of spinal fluid. The patient immediately stopped breathing and could not be resuscitated.

143. Goldberg. S.: Report of Two Cases of Pneumococcic Meningitis; Treated by Intraventricular and Intraspinal Injections of Felton's Antibody Solution and Intraventricular Irrigation; Treated by Intraspinal Injection of Optochin (Ethyl Hydrocuprein Hydrochloride), M. J. & Rec. **129**:219 (Feb. 20) 1929.

Dangers of Ventricular and Cisternal Puncture in the Treatment of Epidemic Meningitis.—Neal [144] stated that she has never seen any immediate harm follow a ventricular puncture. On the other hand, comparatively few babies for whom this treatment has been necessary have recovered. Neal does not believe that the after-effects of repeated trauma to the brain tissue are known at present. She stated:

> As regards cisternal puncture, I do know that death has followed, due to hemorrhage. It seems unfortunate that these fatalities are not reported, as we read accounts of large series of cases without accident written by physicians using this technic on patients with syphilis of the central nervous system. The dangers are much greater, I believe, when there is an active inflammatory process with layers of adhesions in the neighborhood of the cistern, as is likely to be the case in meningitis.

144. Neal, Josephine B.: The Treatment of Epidemic Meningitis, New York State J. Med. **30**:79 (Jan. 15) 1930.

Abstracts from Current Literature

Ear

A Benign Form of Gradenigo's Syndrome. R. Gaillard-Bujadoux and Guichard, Ann. d'ocul. **169**:750 (Sept.) 1931.

Gaillard-Bujadoux and Guichard observed a benign form of Gradenigo's syndrome in a young girl, 18 years of age. Complete paralysis of the external rectus was noted in the course of erysipelas and acute otitis media. The roentgenograms were positive, and antrotomy was performed. The paralysis of the sixth nerve improved rapidly following this operation.

Berens, New York [Arch. Neurol. & Psychiat.].

Temporosphenoidal Abscess on the Left Side, of Mastoid Origin. Drainage by Lemaitre's Method. Castelnau, Rev. d'oto-neuro-opht. **9**:563 (Oct.) 1931.

This case report concerns a woman, aged 45, who had had a suppurating left ear since childhood. Seven days before the consultation, an acute lighting up of the infection occurred, accompanied by persistent headache and occasional attacks of vertigo and otalgia. After another week of observation, an operation on the mastoid was done, but the progress of healing was unsatisfactory. The headaches persisted; the temperature remained above normal, and the wound was filled with unhealthy granulations. No clear signs of intracranial involvement were elicited, and the eyegrounds and spinal fluid were normal. On the ninth postoperative day, the patient suddenly became worse: The lassitude increased, vomiting recurred, and the psychic state was changed, but no other evidence of intracranial mischief was apparent. The urine was scanty; urea was found in the spinal fluid and was increased in the blood. During the night, coma developed. Exploration of the temporal lobe revealed an abscess containing 5 cc. of pus. The abscess was drained by the method of Lemaitre.

The evolution of the abscess probably began in the first days of the acute exacerbation of the mastoiditis. The course of the disease was modified by the partial decompression incident to the first operation. Atypical mastoiditis should suggest the presence of cerebral abscess, and persistent headache is a valuable sign, especially when accompanied by change of character and lassitude, even in the absence of torpor, obnubilation and aphasia. The method of operation on a brain abscess devised by Lemaitre (location of the abscess by needle puncture and drainage by a small rubber tube, increased in size at each dressing, in order to shut off the subarachnoid spaces from contamination) is highly extolled.

Dennis, Colorado Springs, Colo. [Arch. Neurol. & Psychiat.]

Brain Abscess Treated by the Method of Exclusion of the Subarachnoid Spaces. Aubin and E. Baudouin, Rev. d'oto-neuro-opht. **9**:582 (Oct.) 1931.

One more successful result from the method of treatment for brain abscess by exclusion of the subarachnoid spaces is added to the number previously reported.

A man, aged 40, who had otorrhea in the right ear since childhood, was in a precarious condition when first seen, with marked contraction and stiffness of the neck, profound coma, a temperature of 40.5 C. (104.9 F.) and a pulse rate of 130 per minute. No diagnosis other than generalized meningitis was possible. Immediate operation was undertaken. The spinal fluid, taken immediately before

the intervention, was clouded, and no micro-organisms were found on direct examination. When the roof of the antrum was removed, a large extradural abscess was discovered, and the meninges were red and fungoid. Puncture of the temporal lobe revealed two coffeespoonfuls of greenish-yellow, fetid pus. A small rubber drainage tube was substituted for the needle, and the wound was dressed. The pus contained streptococci and gram-positive and gram-negative anaerobic bacilli. Improvement was immediate. The drain was increased progressively in size at each dressing. On the fifth postoperative day an intercurrent infection of the wound caused a rise of temperature. Reexamination of the patient revealed: The stiffness of the neck was still present; the sign of Brudzinski was positive; there was a flaccid right hemiplegia, more marked in the arm, and Babinski's sign was positive. The face was not involved in the paralysis; sensibility was not disturbed, and there were no ocular abnormalities. A striking change in the psychic state of the patient occurred. He had no interest in himself or his surroundings, although his comprehension was perfect. Ordinarily taciturn, he became loquacious, and his conversation was accompanied by immoderate, puerile laughter. There was no interruption of the favorable course of the healing, and the cure was complete in seven weeks. Five months after the operation he was able to resume his occupation.

Attention is called to the unusual seat of the hemiplegia, the signs of purulent meningitis, the psychic alterations, analogous to those seen in patients following an operation for cerebral tumor, and the advantages of the method used for draining a brain abscess.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

PATHOGENESIS AND THERAPY OF LABYRINTHINE ROTATORY VERTIGO. A. GLASSCHEIB, Monatschr. f. Ohrenh. **66**:274 (March) 1932.

After reviewing the mechanism of rotatory vertigo, Glasscheib inquires into the reasons for the occurrence of angiospasm or vasodilatation in rotatory vertigo, especially in the type which is of endocrine origin and appears after emotional disturbances, sexual excitation and during menopause. This type is sympatheticotonic, alkalotic or vasoconstrictor. The author states that recent work indicates that there is an atrophy of the suprarenal cortex during menopause and a consequent cholin deficiency of the blood is to be expected. (Investigations of cholin content of blood in vestibular vertigo are being conducted.) This deficiency of cholin, which is a vagotropic hormone, results in a preponderant influence of the sympathetic nerve and therefore in vasoconstriction. In emotion or sexual excitation, the preponderance of sympathetic influence due to secretion of epinephrine is to be expected. Therapy of sympatheticotonic vertigo consists of prevention of vasoconstriction by administration of cholin and prevention of endothelial secretion by injection of free iodine in minute quantities.

EDITOR'S ABSTRACT.

PATHOLOGY OF SECRETORY CATARRH OF THE EAR. E. SCHLANDER, Monatschr. f. Ohrenh. **66**:278 (March) 1932.

Schlander reports his study of the secretion of the middle ear in so-called secretory catarrh conducted for the purpose of determining whether it is an exudate or a transudate. He made a chemical analysis of the secretion in sixteen typical cases to determine the total protein content, using the micro-Kjeldahl method according to Preger. The specific gravity was computed according to the formula of Ruess. According to these values, there were nine exudates and five transudates, while two cases were on the borderline. The author found that in all fresh cases of secretory catarrh developing from acute rhinopharyngitis the secretions were exudates of high protein value. In cases in which the catarrh had existed a long time and there were no acute changes in the nasopharyngeal mucous membrane, the secretions had the low protein values of transudates. Only two of the five cases with transudates, namely, those with

tumors of the epipharynx, could be considered of purely mechanical causation. The possibility of a change from exudate to transudate and several factors pointing to such an hypothesis are discussed.

EDITOR'S ABSTRACT.

LATE COMPLICATIONS FOLLOWING RADICAL OPERATION IN OTITIS MEDIA. M. TAMARI, Monatschr. f. Ohrenh. **66**:286 (March) 1932.

From a study of the records of operations of the last thirty years, Tamari reports that in 3,273 radical operations on the middle ear performed in the last two decades, there have been only 11 cases of late endocranial complication. The reports of these 11 cases are given. The author considers radical operation in chronic otitis media primarily as a prophylactic operation against endocranial complications. The fact that in 11 cases patients suffered endocranial complications many years after the radical operation teaches that the sinus resulting from radical operation requires special attention in cases of persistent secretion or acute exacerbation, but it does not deny that the operation, so performed that all existing cells are removed and a cholesteatoma is completely cleansed, constitutes a preventive measure against complication. The exceptions concern either radical operation performed during the acute stage of otitis, incomplete operations or operations in which, though carried to completion, conditions such as atresias, tissue bridges or scar formation, which present an opportunity for retention or renewed infection, were established. In general, one can say that the radical operation with persistent, large retro-auricular opening which facilitates an exact oversight of the surgical cavity and in which wound conditions can be so influenced by postoperation therapy that neither bridges nor neoplasm can form have always given protection against endocranial complications. The author mentions further that, although the number of cases is few, he did not receive the impression that exposure of the sinus and the dura by operation affects the prophylactic value of the radical operation.

EDITOR'S ABSTRACT.

IONIZATION WITH ZINC AFTER FRIEL: ONE OF THE METHODS OF THERAPY IN CHRONIC OTORRHEA. A. F. PUSCHKIN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:329 (Feb. 29) 1932.

Puschkin reports the successful treatment of chronic otorrhea by ionization with zinc according to the method of Friel. He describes in detail the technic employed, which contained some modifications of Friel's method. The author thinks that ionization has several important advantages over other methods. The zinc penetrates the diseased tissue and forms a coagulum on the surface, but it does not affect the tissues that are cicatrized or in the process of cicatrization. The medicament penetrates gently into the entire affected part, which in most cases is not accessible to other methods of treatment, and so the newly formed ions have a deeper effect on the diseased tissue than is possible with other methods. The galvanic current simultaneously exerts a favorable action on the tonus of the healthy adjacent tissue. The rapidity of the method, as to both treatment and healing process, is emphasized. The author thinks that this method deserves the attention of the otorhinologists and that it belongs in the front rank of methods of therapy in chronic otitis because of its rapid effect and good results; the percentage of cures, even in neglected cases, reaches 80 per cent. The easy acquisition of simple, cheap apparatus makes the method accessible to every specialist, and it can be used for ambulant treatment as well as in hospitals.

EDITOR'S ABSTRACT.

CONSTITUTION IN OTOSCLEROSIS. O. MUCK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:375 (Feb. 29) 1932.

Since 1925 Muck has performed the epinephrine probe test on all of his patients who were afflicted with otosclerosis, and he has found the pathologic, vasomotor

reflex phenomenon of the white streak in every case. This indicates that the tonus in the region of the internal carotid artery or the auditory artery is disturbed in these cases. The innervation of the auditory artery by fibers of the sympathetic system has been recently demonstrated. As the close relation between the endocrine system and the vascular system is known, the author thinks that this gives a key to further study. Recent investigations indicate that there is a disturbance of intermediary metabolism in otosclerosis, and some investigators attribute this in part to endocrine causes. The author was able to demonstrate by means of a change of diet that there are metabolic disturbances in persons afflicted with otosclerosis. A meatless diet was administered for five days to thirty-seven women and girls suffering from otosclerosis; at the end of this period the white streak sign either disappeared entirely or the duration of contraction of the white streak was decreased to about one half of its length previous to the institution of the meatless diet. On renewed consumption of meat the white streak sign reappeared. The author thinks that it would be valuable to make a study of the metabolism of patients with otosclerosis before and after administration of the diet described by him.

EDITOR'S ABSTRACT.

CEREBRAL SYMPTOMS IN UNCOMPLICATED OTOGENOUS EXTRADURAL ABSCESS AND IN SINUS THROMBOSIS. W. UFFENORDE, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:151 (Feb.) 1932.

Uffenorde reports two cases of uncomplicated otogenous extradural abscess and three cases of uncomplicated sinus thrombosis with cerebral symptoms. In calling them uncomplicated, he means that the changes that caused the cerebral symptoms were only incidental to the main disease and tended to recede spontaneously after intervention in the latter; from an anatomicopathologic standpoint, they were not uncomplicated. The symptoms were foot-clonus, Babinski's reflex and marked bilateral hypotonia, all of them distinct and persistent. The first case was that of a boy, aged 10, with chronic suppuration of a cholesteatoma on the left side, who had an extradural abscess in the posterior cranial fossa. The pronounced Babinski's reflex on the right side was still present four weeks after recovery from the operation. The second case was that of a man with acute retrotympanal otitis media and an extradural abscess in the posterior cranial fossa on the right side. There was a pronounced foot-clonus on the right side and a temporary Babinski's reflex. The clonus was still distinct four weeks after dismissal of the patient. The third case was one of otitis media with cholesteatoma, large perisinuous abscess and extensive sinus thrombosis. After elimination of the thrombosed sinus, a pronounced foot-clonus appeared contralaterally, lasting about one month. The fourth case was that of a sinus thrombosis following acute mucosus otitis. There was a Babinski's reflex on the contralateral side, which was still present more than a year after the operation. The last case was that of a sinus thrombosis following cholesteatomatous suppuration with unusual extension of the sinus changes to the other side, with metastases and a large lung abscess. This case exhibited marked bilateral hypotonia of the lower extremities. The case is of special interest because the symptoms of hypotonia, nystagmus to the diseased side and retarded and scanning speech without labyrinth disturbance superceded the septic symptoms and pointed to a cerebellar abscess. Opposed to this view was the fact that the disturbance was on both sides, and this strengthened the author in diagnosing the cerebellar aspect of the complex clinical picture as a collateral infectious-toxic and local manifestation of stasis in the lymph and blood circulation in the cerebellum due to disintegration of the thrombus and venous influxes. The author thinks the most important conclusion to be drawn from these cases is that the symptoms of Babinski's reflex, foot-clonus and pronounced hypotonia may accompany extradural abscess and sinus thrombosis without the existence of a serious cerebral process which demands specific therapeutic measures.

EDITOR'S ABSTRACT.

SIGNIFICANCE OF GENERAL METABOLIC DISTURBANCES IN OTOSCLEROSIS. K. GRAHE and C. GRIEBEL, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:162 (Feb.) 1932.

Grahe and Griebel think that in otosclerosis there is a primary change of the blood serum which results in increased decalcification of the labyrinth bone; the labyrinth bone is the only one affected because it differs from the rest of the skeleton in chemical composition. This opinion is based on a series of experimental studies of the relation of bone decomposition and calcium content of the blood serum. The authors first studied the composition of various bones and found a higher calcium and phosphorus content in the labyrinth bones than in the other bones of the body. A comparative study of the decalcification of the labyrinth bone and the temporal bone was made by the addition of blood serum to pulverized bone and gravimetric determination of the calcium content of the filtrate following incubation. The calcium decomposition of the labyrinth bone and the temporal bone by normal blood serum was practically the same, but when the serum of otosclerotic patients was used the decalcification of the labyrinth bone greatly increased compared to that of the temporal bone. The blood serum of otosclerotic patients was found deficient in calcium. In all cases the decalcification of the labyrinth bone was inversely proportional to the calcium content of the serum, and decrease of the calcium content of the serum below normal was accompanied by strong increase in decalcification of the labyrinth bone.

EDITOR'S ABSTRACT.

Pharynx

TONSILLECTOMY VERSUS MEDICAL TREATMENT IN CASES OF RHEUMATIC FEVER. WILLIAM H. ROBEY, M. Clin. North America **15**:875 (Jan.) 1932.

Tonsillectomy is an efficient means of stopping an attack of acute rheumatism when the usual purely medical measures have failed. The operation, even in the presence of fever and inflamed joints, is not more dangerous than interval tonsillectomies. It should not be done while active acute tonsillitis is present, but this question should seldom arise, since acute rheumatism is usually a sequel of acute tonsillitis. The operation should be performed only by those having received special training. It is important to remove thoroughly all tonsillar tissue.

SMITH, Ogden, Utah. [AM. J. DIS. CHILD.]

BACTERICIDAL POWER OF BLOOD DURING TONSILLITIS. W. VOGEL, Deutsches Arch. f. klin. Med. **172**:298 (Dec. 21) 1931.

During the course of every case of acute tonsillitis, the bactericidal power of the blood decreases, and it increases slowly with the clinical improvement of the patient. With one exception, none of Vogel's patients had complications.

DOOLEY, Kent, Conn. [AM. J. DIS. CHILD.]

AN ENDEMIC OF TONSILLITIS IN ALPS: CONTRIBUTION TO ETIOLOGY OF INFECTIOUS DISEASES. M. WASSERMANN, Monatschr. f. Ohrenh. **66**:144 (Feb.) 1932.

Wassermann reports an endemic of tonsillitis in an Alpine hotel in December in which twenty-six of a total of eighty or ninety guests were stricken. All but one of the cases were in women and children. There were no cases among the hotel staff. While the infection may have been spread by the room servants, the selectivity of the endemic infection can be explained only on the basis of individual predisposition. This predisposition is determined in part by hereditary factors, such as anlage of the connective tissue and constitutional resistance of the blood and in part by the acquired state of health and nutrition which is in turn affected by local and temporal influences. Exposure to the increased heat and

light radiation of the sun in Alpine climates, and especially to the chemically active short wave length rays, results in increased metabolism. This is marked at first by increased decomposition of body proteins and may result in an inflammatory disposition of the tissues which, coupled with the increased protein decomposition resulting from sudden strenuous and unaccustomed activity in sports and possible lowering of resistance from overexposure to cold and moisture, predisposes the individual to infection. This accounts for the selection of women and children who, either because they were there for convalescence, or because they were less used to sports than men, easily overtaxed themselves. For prophylaxis, the author advises that young or physically weakened persons do not make the sudden transition from low elevation to Alpine heights, but accustom themselves to the sun's rays by a stop at an elevation of about 1,000 meters. Initial exposure to the sun's rays should be brief, and all physical exertion should be avoided during the first days of acclimatization. The author thinks this report illustrates the importance of individual predisposition in the incidence of disease and of the effect of external and internal influences on this predisposition.

EDITOR'S ABSTRACT.

TRAUMATIC TONSILLITIS AND LIABILITY OF THE PHYSICIAN. M. GOERKE, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:129 (Feb.) 1932.

Goerke reports a case of traumatic tonsillitis, which is of medicolegal interest, and makes it a point of departure for a discussion of the incidence, pathogenesis, prophylaxis and prognosis of traumatic tonsillitis. In the case reported, traumatic tonsillitis followed septum resection. A few days after the patient had been dismissed, an acute otitis media developed, and his 5 year old son was stricken with scarlet fever. The patient refused to pay the bill of his rhinologist and presented him with a counter bill for the expense incurred by his son's illness, based on the claim that the rhinologist had not warned him that 90 per cent of endonasal operations are followed by tonsillar infection, and that further he had not been warned of the contagious nature of the tonsillitis and consequently had let his son kiss him when he came home. In his discussion of traumatic tonsillitis, the author says that statistics on the incidence are so at variance that he thinks the method of operation and the postoperative therapy must have something to do with the occurrence of tonsillitis. As to pathogenesis, the author thinks there is a transmission of infectious material from the nose to the tonsils probably over the lymph channels. He considers it unquestionable that nasal tamponade is largely instrumental in causing an infection, as it obstructs the natural drainage of the wound secretions, inflammatory products and tissue débris, and their removal by the lymph channels is favored. As to prophylaxis, the first requirement is utmost asepsis even in the most insignificant nasal operation, and the second is avoiding tamponade whenever possible. It cannot be omitted in submucous septum resection, but it can be omitted after extraction of polyps and concha resection. Prognosis is usually favorable.

EDITOR'S ABSTRACT.

TONSILLECTOMY IN CHILDREN. F. REUTER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:186 (Feb.) 1932.

Reuter presents his reasons for preferring tonsillectomy to tonsillotomy in children and discusses tonsillectomy in children from the standpoint of indication for operation, technic and results. The author observed that most adults who came to him for tonsillectomy had undergone tonsillotomy when they were children. The complaints for which tonsillotomy was undertaken often recurred more severely after the operation. Disease of the cervical glands in adults was often cured by tonsillectomy, and the author assumed a similar reaction in children. In children in whom the tonsillotomy was practically a tonsillectomy, results were especially favorable. These and other reasons led him to the exclusive use of tonsillectomy in children. The indications for tonsillectomy are: (1) hyper-

trophy of the tonsils, if they are constantly pathologic; (2) chronic tonsillitis with or without hypertrophy, and (3) all cases of recurring tonsillogenous disease. The author performed tonsillectomy in 547 children, without any injuries resulting from operation. The susceptibility to colds and other disease was decreased. Great improvement in appetite, growth and nutrition was observed. A good effect on diseases of the cervical glands was observed. Disturbances of speech were not observed. He describes his method of operation. Local anesthesia is always employed. He does not use a gag, as this causes the children to scream and press, resulting in increased hemorrhage. The importance of understanding child psychology is stressed. He has had no hemorrhages and thinks these are more likely to occur in tonsillotomy than in tonsillectomy. Tonsillectomy in children is not followed by functional deficiency, because only a part of the lymphatic ring of the pharynx is removed in extirpating the palatine tonsil. In children the regenerative capacity of the tissue is strong enough to form new tissue if too much has been removed, thus protecting the child from injury.

EDITOR'S ABSTRACT.

Nose

ETHMOIDITIS IN CHILDREN. S. P. SCHECHTER, Arch. Pediat. **49**:115 (Feb.) 1932.

Attention is called to the frequency of ethmoiditis in children and its usual symptoms. The frequency with which sinus diseases are overlooked is deplored. Failure to diagnose these conditions properly is perhaps due to the fact that roentgenology as a means of diagnosis is not so reliable in young children as in adults. Transillumination is frequently unsatisfactory, and subjective symptoms are not well expressed. Frequent colds, with nasal discharge or obstruction, should arouse suspicion of sinus disease. Irritability, anorexia, anemia, headache, irregular temperature, recurring adenitis and chronic bronchitis are signs that one has to consider in diagnosing sinus disease in children. In diseases of the joints, when the tonsils and adenoids are removed and there is no cessation of arthritic symptoms, sinus disease must be seriously considered. Four case reports are given, and the usual methods of treatment discussed. Diseased tonsils and adenoids are believed by the writer to cause 80 per cent of the cases of sinusitis in children.

WADDELL, JR., University, Va. [AM. J. DIS. CHILD.]

SINUSITIS IN CHILDREN. WARREN B. DAVIS, Pennsylvania M. J. **34**:305 (Feb.) 1931.

The author cites anatomic considerations attendant on the development of the paranasal sinuses in early life and states that from earliest infancy sinusitis is a condition to be considered clinically, occurring in the different sinuses in descending order (ethmoid, maxillary, sphenoid and frontal). Two groups of predisposing etiologic factors are enumerated: (1) local conditions interfering with nasal ventilation and drainage, enlarged infected tonsils and adenoids, swollen mucosa from allergic conditions, anatomic defects and malformations, foreign bodies and tumors, and (2) lowered general resistance from poor hygiene, unbalanced diet, metabolic disturbances and endocrine disorders. The condition occurs also as a sequel to measles, scarlet fever, acute bronchitis, pneumonia, pertussis and diphtheria. Among the diagnostic methods, transillumination is of less value than correctly interpreted roentgenograms. As most cases of acute and subacute sinusitis respond to general and local treatment, beyond the removal of infected tonsils and adenoids, surgical measures should be employed conservatively, should be preceded by careful study of roentgenograms and should be marked by good judgment, accurate anatomic knowledge and refined surgical technic.

HALL, Uniontown, Pa. [AM. J. DIS. CHILD.]

AN ATYPICAL SYNDROME OF THE NASAL NERVE. CARLOS CHARLIN, Ann. d'ocul. **168**:808 (Oct.) 1931.

The syndrome of the nasal nerve previously reported on by Charlin (*Ann. d'ocul.* **168**:86 [Feb.] 1931) is now considered by him in its atypical form. In this paper he states that neuritis of the nasal nerve may occur in an incomplete and atypical form. In some cases the symptoms are primarily ocular; in others, the nasal symptoms predominate. In the latter type there is unilateral, oculo-orbital pain, and the most careful examination of the eye is required to detect tenderness of the eyeball, slight hypotension or superficial keratitis. In some cases a mild cyclitis is discernible only with the slit-lamp. The nasal mucous membrane is hypersensitive, congested and moist in the areas supplied by the nasal nerve. Applications of cocaine and epinephrine to the nasal mucous membrane in this area bring about rapid amelioration. The diagnosis is often facilitated by palpating the cutaneous distribution of the nasal nerve. If these points are painful, a nasal examination should be ordered.

BERENS, New York [ARCH. NEUROL. & PSYCHIAT.].

CONTINUOUS DRAINAGE FOLLOWING OPERATION ON THE FRONTAL SINUS. A. SEIFFERT, Monatschr. f. Ohrenh. **66**:67 (Jan.) 1932.

Seiffert states that the success of operations on the frontal sinus in which the cavity is preserved depends on keeping the passage that connects it with the nasal cavity continuously open, and prohibiting the shrinking process. The author describes a drain that can be introduced by the patient, which does not exert a strong pressure or irritate the tissues and remains fixed in place. The drain described consists of a club-shaped pipet suction cap of fine transparent rubber, which is provided with several large openings laterally near the fundus. The lower rim is cut off and a little thread attached to the cap. This cap, after being lubricated, is stretched over a nasal sound by pulling the thread and is thus introduced into the surgically enlarged frontal sinus. When the thread is released, the cap widens and is thereby kept in place. The drain can be left in the passage several days; for removal, it is only necessary to pull the thread with forceps. The drain comes out readily because of its softness, but sensitive patients may be lightly anesthetized before changing of drains.

EDITOR'S ABSTRACT.

TONICITY OF SYMPATHETIC NERVOUS SYSTEM AND CONTENT OF CALCIUM AND POTASSIUM IN BLOOD SERUM OF PATIENTS WITH ATROPHIC RHINITIS, OZENA AND SCLEROMA. A. W. WIRABOFF and S. A. TERIAN, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:116 (Dec.) 1931.

Wiraboff and Terian report that the calcium content and the potassium content of the blood serum of patients with atrophic rhinitis, ozena and scleroma are in most cases within normal limits. These diseases evidently do not cause a characteristic change in the ionic equilibrium of the blood. Equilibrium disturbances of the tonicity of the sympathetic nervous system in patients with atrophic rhinitis, ozena and scleroma were observed, but no definite conclusions could be made as to which part of the sympathetic system was affected.

EDITOR'S ABSTRACTS.

Miscellaneous

BRONCHOSCOPY IN CHILDREN. ANDRÉ BLOCH and A. SOULAS, Bull. Soc. de pédiat. de Paris **29**:398 (July) 1931.

The authors review the indications for bronchoscopy in children, e. g., foreign bodies in the bronchi, bronchial obstruction due to papillomas, congenital stenosis, inflammatory stenosis and bronchopulmonary suppuration. The narrowness of the epiglottic opening is discussed, and the question of the advisability of performing

tracheotomy and then "inferior" bronchoscopy, as opposed to "superior" bronchoscopy, which has in some instances been followed by subglottic edema, is mentioned. The latter condition need hardly occur if an instrument of the proper size for each particular case is used, and the bronchoscopist's technic is good. The conclusion reached is that peroral bronchoscopy without any anesthesia, general or local, in the child, is not a dangerous procedure in proper hands, and should be used whenever it is indicated.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

MEDICAL DIATHERMY IN TREATMENT OF OTORHINOLARYNGOLOGIC DISEASE. H. SCHAEFER, Ztschr. f. Laryng., Rhin. (Teil. 1: Folia oto-laryng.) **22**:218 (Feb.) 1932.

Schaefer discusses the use of medical diathermy which he calls conservative diathermy in otorhinolaryngology. The physiologic effect of medical diathermy in otorhinolaryngologic therapy is discussed, also the technic of application (types of electrodes, application of electrodes, strength of current and duration of treatment) are described. The indications for diathermic therapy in otorhinolaryngology are reviewed. Favorable results may be expected in subacute and chronic diseases of the throat such as pharyngitis, laryngitis and tonsillitis, in eczema or furunculosis of the auditory meatus, chronic otitis media and in thyrotoxicoses. Relatively good results were observed in otosclerosis and chronic progressive baryecoia, whereas in diseases of the nose (with exception of acute rhinitis) and of the nasal sinuses, the therapeutic value is slight. The author thinks the therapeutic effect of medical diathermy can sometimes be increased by synchronous use of other therapy such as internal medication, galvanization, roentgenization, application of surface heat, etc. In some refractory cases an optimal effect is obtained in this way. Contraindications for medical diathermy are pointed out. The author thinks that the results obtained show that diathermy is deserving of interest as a therapeutic factor in otorhinolaryngology and justify the assumption that development and perfection of the method will increase the scope of indications for its use and heighten its therapeutic effect.

EDITOR'S ABSTRACT.

ROENTGENOLOGIC DEMONSTRATION OF OTOGENOUS BRAIN ABSCESSES. L. KRAUS, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:230 (Feb.) 1932.

Kraus discusses the roentgenologic visualization of brain abscesses by filling the abscess with an opaque medium and reports four cases of brain abscesses in which he used the method instituted by Mayer, with some modifications. In a preliminary discussion of drainage the author says that choice of method is not important if effective drainage is maintained and retention prevented. The author found that tamponade of the abscess cavern with gauze soaked in 50 per cent methiodal solution was the most effective procedure for roentgenologic visualization. Methiodal is more fluid than other substances used for this purpose, therefore it penetrates better and does not run out as quickly. By employing tamponade of the entire cavity instead of plugging the opening of a cavity filled with fluid, the danger of the fluid seeping out and producing confusing shadow spots is removed. When the filling is not complete, the retained air reveals the size of the cavern. This is most successful when photography is undertaken immediately after the first drainage. When proper care is exercised and the tamponade is carried out slowly, this method presents no dangers, and in one case a striking improvement was observed. The author thinks it desirable that this method of filling otogenous brain abscesses be developed as its use not only makes possible the visualization of the size and depth of the abscess but also gives an insight into the healing of such abscesses.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, April 4, 1932

HOWARD C. BALLENGER, M.D., *President*

RADIOTHERAPY OF CANCER OF THE TONSIL, PHARYNX AND LARYNX. DR. MAX CUTLER (by invitation).

Thirty years of investigation have yielded important physical, pathologic, biologic and clinical data, a correlation of which has served to place the radiotherapy of malignant disease on a sound scientific basis. By far the most important advances in radiation therapy have occurred during the last five years. The late clinical results of the newer technics are just beginning to appear.

Estimation of Radiosensitivity.—The degree of radiosensitivity of a tumor may be estimated after a consideration of three kinds of factors: clinical, histologic and radiotherapeutic.

1. Clinical experience has taught that some organs are more often the seat of radiosensitive tumors than others. The anatomic site of a tumor, therefore, constitutes the first clue as to its probable radiosensitivity.

2. Epidermoid carcinomas of the skin and mucous membranes are radiosensitive, whereas adenocarcinomas, melanomas, fibrosarcomas and osteogenic sarcomas are highly radioresistant.

3. The response of a tumor to radiation (the therapeutic test) remains the most accurate and certain clue to its radiosensitivity. The tumor bed is an important factor in radiosensitivity. Thus the primary lesion may respond promptly to radiation, whereas the metastasis in regional lymphatic glands may fail to regress after much more intensive radiation. A highly radiosensitive tumor situated in lymphatic glands may disappear rapidly after radiation, and the same kind of lesion situated in the liver may fail to respond.

Two important advances have been made during the last few years in the treatment of cancer of the tonsil, pharynx and larynx:

Coutard of the Curie Institute (Paris) has developed a technic of radiation in which the entire scheme of therapy rests on a biologic basis. Forty-two patients irradiated with roentgen rays during the years from 1920 to 1926 for epitheliomas of the tonsil, hypopharynx and larynx and now free from symptoms were treated by the method called "epidermicide." All presented "radio-epithelitis" of the mucous membrane and "radio-epidermitis" of the skin. When the duration of irradiation was reduced below from thirteen to fifteen days, as for example, from five to seven days, a recurrence always resulted. In order to obtain a typical radio-epithelitis it is necessary that the mucosa receive a dose equal to 35 Holzknecht units (3,500 roentgens) or a total cutaneous dose of approximately 60 Holzknecht units (6,000 roentgens).

Coutard reported the following interesting results in epitheliomas of the tonsillar region, hypopharynx and larynx treated with the roentgen rays between 1920 and 1926:

Proportion of Survivals at Beginning of 1931

	Patients Treated	Symptom Free, 1931	Per Cent "Cured"
Males	197	33	16
Females	15	9	60
Total	212	42	20

The epitheliomas of the tonsillar region, in spite of the extensive primary growth and metastases to the cervical nodes, have yielded good results. Among the patients treated between 1921 and 1926, 23 per cent were well at the beginning of 1931. Since 1926, the results have shown an appreciable improvement (46 per cent).

A critical analysis of the technic employed by Coutard clearly indicates one important factor that characterizes this method. The flow of energy of the roentgen rays is slowed to approximately 250 roentgens per hour. The patient receives from twenty-five to thirty-five hours of irradiation extending over approximately fifteen days. This distribution permits the delivery of a formidable dose of radiation into the center of the tumor (approximately 3,500 roentgens). The unusually large dose and the technic of prolonged irradiation constitute the important factors in the result.

Another important advance in radiotherapy has been reported from the Radiumhemmet (Stockholm). In a most thorough and comprehensive monograph on malignant tumors of the tonsil, Berven compared the results of radiotherapy of these lesions with the old technic and the new technic.

Among twenty-eight patients with carcinoma of the tonsil treated between 1919 and 1924 with roentgen rays and small doses of radium (old technic) primary healing occurred in 25 per cent. At the end of one year 10 per cent were free from disease; at the end of two years all the patients had had recurrences.

Among eighteen patients with carcinoma and lympho-epithelioma of the tonsil treated between 1923 and 1927 by the new technic with large doses of radium at a distance (teleradium-radium "bomb") seven patients, or 39 per cent, were free from disease three years after treatment.

A comparative study of these results and the methods employed leads to the obvious conclusion that the results of radiotherapy in malignant disease depend not only on the extent of the disease but to a pronounced degree on the method and technic of irradiation. The important factors are: (1) the total dose; (2) the total time of irradiation; (3) the correct division, distribution and duration of individual exposures, and (4) the quality of the irradiation.

As to the choice between the use of roentgen rays and those of large quantities of radium at a distance the latter method offers important advantages.

First, it should be noted that the "Coutard method" constitutes an attempt to imitate radium by means of roentgen rays. The flow of energy from the source of radiation is slowed so as to permit a prolonged irradiation. In order to accomplish this Coutard increases the distance and the filter, and reduces the amperage, making it possible to treat the lesion for from twenty-five to thirty-five hours.

Berven stated that the reaction of the normal mucous membrane produced by large doses of radium (radium epithelitis) causes less local subjective disturbance than that produced by roentgen rays (roentgen epithelitis). He also reports that the general reaction of the body is as a rule slight as compared with "roentgen sickness" so frequently seen after treatment with large doses of roentgen rays. Cardiovascular disturbances reported by Coutard after roentgenotherapy were entirely absent in Berven's patients treated by the radium pack.

In a report before the Third International Congress of Radiology, in July, 1931, Forssell stated that in the treatment of tumors radium is superior to roentgen rays in most instances. The general disturbance of the patient is less marked, and late effects of radiation are less common. This is particularly true in the treatment of cancer of the buccal cavity and pharynx, and also in metastases to the lymph nodes and in cancer of the breast. Forssell concluded that the use of large quantities of radium at a distance constitutes the most important advance in radiotherapy since Dominici first taught the use of ultrapenetrating rays.

In the tumor clinic of the Michael Reese Hospital I employ 4 Gm. of radium in the form of a radium "pack" or radium "bomb" (teleradium) in the treatment of cancer of the tonsil, pharynx and larynx. Metastases to the lymph nodes are also treated in this manner. The apparatus constructed in collaboration with

Dr. Failla was designed to offer the maximum protection to the normal tissues surrounding the growth and to the body in general. Three portals of entry are available (5, 7 and 10 cm. in diameter). The smallest portal (5 cm. in diameter) was designed especially for the treatment of early localized carcinomas, particularly those of the larynx. The principles of the technic employed are similar to those of Coutard except that radium is employed instead of roentgen rays. By this means an effort is made to duplicate the remarkable results of Coutard with milder local and constitutional reactions than is possible with roentgen therapy.

DISCUSSION

DR. RICHARD H. JAFFÉ (by invitation): In the time allotted to me I can take up but a few of the most important questions concerning the action of radiating energy on tumors. The first of these questions deals with the morphologic changes that the tumor cells undergo after they have been irradiated. Two days after the irradiation practically all of the mitotic figures have disappeared, and one can judge from this finding that the most sensitive part of the tumor is the mitoses. Two days later mitoses again become visible, but they show now marked signs of degeneration, many of the chromosomes being broken up into irregular granules. The next step is the disintegration of the tumor cells. The time interval between irradiation and the onset of the disintegration varies greatly, and it may be from a few days to more than two weeks before the regressive changes become manifest. The nuclei of the tumor cells swell, become vacuolated and segmented and finally disappear. The cytoplasm loses its granular structures and appears homogeneous or vacuolated, and polymorphonuclear leukocytes invade the cells to destroy what is left of them. Some débris may also be taken up by macrophages. At the same time the stroma starts to proliferate and gradually replaces the breaking down tumor cells. None of these changes, however, is specific of the action of radiating energy, as all of them occur spontaneously also. The phenomena are merely an exaggeration of what is seen in any malignant, fast-growing tumor.

One has to keep in mind, however, that even after massive doses of irradiation, islands of intact tumor tissue may remain alive in the necrotic masses, islands that give rise to recurrent growths that not seldom prove to be much more radioresistant than the original tumor. The necrotic tissue in itself may be harmful to the patient. Since not only by resorption do toxic split products enter the circulation but the necrotic tissue exerts a stimulating influence on remaining tumor cells, inciting a more rapid growth (Caspari's necrohormones). In addition, radium and roentgen rays act on the body as a whole, stimulating and eventually depressing its defense reactions, a fact that is often neglected in modern radiotherapy. The stimulation of the entire active mesochyma (reticulohistiocytic system) is unquestionably a very important factor in the curative effect of the radium or roentgen rays. Much harm, however, is done, if the dosage is such as to depress rather than to stimulate the mesenchymes.

As Dr. Cutler has pointed out, there is a certain parallelism between the histologic structure of a tumor and its radiosensitivity. The less mature the tumor cells, the greater is their sensitivity to radium and roentgen rays. This rule can be applied, however, only with a certain reservation. Tumors of identical histologic structure may respond differently to the treatment, and this again indicates the great importance of the tumor host for the success of the radiation. It has been shown that experimental tumors in animals that differ greatly as to their radiosensitivity lose this difference when grown in vitro. Thus it seems that outside the body all tumor cells react in about the same manner and that the individual differences are the result of the interaction between the body and the tumor.

The malignant epithelial tumors of the nasopharynx are usually subdivided into transitional cell carcinomas and squamous cell carcinomas. The former are less mature, do not show any tendency to hornification and usually respond promptly to irradiation. The latter are characterized by hornification and are

much less influenced by radium or roentgen rays. A third type of tumor, which is questioned by some investigators, is the lympho-epithelioma. It consists of a reticulum of squamous epithelial cells, the meshes of which are filled by lymphocytes. Thus it resembles the thymus. This type of tumor, too, shows marked radiosensitivity. A number of authors identify the lympho-epithelioma with the transitional cell carcinoma, while others consider it as grade III and IV of the squamous cell carcinoma. I believe, however, that the lympho-epithelioma represents a specific type of mixed tumor which is found especially in the region of the tonsils, nasopharynx and thymus. At biopsy it may be difficult to distinguish this tumor from Hodgkin's granuloma because of a certain cellular pleomorphism and the large pale cells with the vesicular nuclei and delicate cytoplasm.

Dr. Cutler has pointed out that radiotherapy is in the stage in which it emerges from experimentation into general use. This stage is the most critical in the development of any therapeutic procedure and requires special criticism in order to avoid disappointment as well as premature discarding. Any further progress can be expected only from close cooperation between the histopathologist, the radiologist and the clinician.

Dr. Joseph C. Beck: I should like to relate a horrible story, covering a good many years, of cases under observation by me and my assistants in which the patients were treated by experts in both roentgen and radium therapy, in order to show why some of the patients died in consequence of insufficient irradiation, or even of insufficient surgical intervention. Although it is a terrible acknowledgment, we did not know any better. We asked the advice of experts, and deep roentgenotherapy was thought to be the treatment that would save our patients. One after another succumbed, if not to the carcinoma, to hemorrhage from the carotid arteries and other large vessels. As both the speakers have pointed out, a change has come in that the radiologist must be an anatomist and a histopathologist, because on that combination of experience the treatment is based.

About five years ago for three weeks I watched Dr. Berven, of Stockholm, work; at that time he was beginning to show the change in the irradiation of the tonsils. He has given a remarkable statistical report. Recently Zuppinger, of Zurich, Switzerland, has published a book in which the opposite results are shown from the use of irradiation in malignant conditions about the pharynx, the tonsils and laryngopharynx; he reported unfavorable statistics and high mortality. These reports are of value in this work in the way that has been shown here, in demonstrating that one must have an expert to tell one just how to apply the dosage and what it should be. Dr. Cutler has shown, I think, to all of us present at this meeting that distant therapy with the radium pack or a large amount of radium is the best method.

I wish to ask Dr. Cutler how he can answer the London letter that appeared two weeks ago in *The Journal of the American Medical Association* (**98**:1096 [March 26] 1932) in which it was stated that physicians who were treating with a large amount of radium had to give it up. The correspondent stated that the radium pack, or the radium bomb, was to be given up. Radiologists have put great hope in that type of treatment, and I wish to know what Dr. Cutler thinks about this report. The methods that have been found successful by my associates and me have been those that Dr. Cutler says cannot be properly gaged, namely, the interstitial application of radon. Through Dr. Larkin's advice and assistance, we have applied these seeds as they should be placed. In treatment of the nose and throat the laryngologists are in a position to apply the treatment, unless it is the distant therapy. We can do a direct laryngoscopy and introduce the direct method of interstitial irradiation, and I think there have been excellent results from this method even though not in a large number of cases. When radon is used there has been considerable difficulty from oozing hemorrhages, with irritative symptoms, and this is the disadvantage. I was compelled to do a ligation of the carotid while Dr. Strauss was giving a transfusion in a case in which implants were used and there was sloughing. The end-result, however, was good.

I should like to hear a little about nonmalignant tumors, and I hope that Dr. Cutler will say something about the vascular tumors that are not malignant, and what is the action of their destruction.

DR. T. C. GALLOWAY: About six years ago I discovered the advantage of electrosurgery in malignant conditions, and with it, I am sure, my associates and I have been obtaining better results than with any method we had before, but we have had a hard fight, and only occasionally in tumors of the pharynx do we have the thrill of winning a desperate battle. Dr. Cutler can show a much higher percentage of five year cures with radium in these tumors. We should be very much encouraged to find that these tumors of the nasopharynx, the tonsil and the base of the tongue in the treatment of which we have been almost helpless are amenable to irradiation, and we shall gladly turn them over to the expert in radiology. That will leave us time and energy to treat other kinds of cancers in which we can get better results.

Thanks to Professor Jaffé, we have been learning what tumors are not susceptible to diathermy or surgery. We are thankful to have the matter explained for us as well as Dr. Cutler has done, and I am sure we shall profit greatly.

If all of the members of this society could come to the County Hospital and see the patients on whom radium therapy has been unsuccessful, to their loss and pain and distress, they would wish that a talk like this could be broadcast to every general physician. If any lesson is to be learned from such a talk it is that there is no place for the casual or occasional user of radium. Radium has occasionally been a too easy method of exploitation and negligence. We have had patient after patient who had inadequate or improper irradiation who has been carried past the period when he could easily have been cured by surgical intervention, diathermy or any other method. Radium must be used not only by a person who knows how to use it but by one who has it in sufficient amounts and can properly gage his treatment by the histopathologic picture of the tumors and by experience.

Dr. Cutler has given us a picture of an ideal situation, but I think that there is an economic aspect. How many of the thousands of persons with potential carcinoma among charity patients in this city are going to be treated properly? Must we who cannot get adequate radium for our charity patients still carry on as best we can with other means?

I think that Dr. Cutler, by pointing out types of tumors that are susceptible to radium, has also shown others that need surgical treatment—and to some of the members of this society who have used it that means electrosurgery.

Another question is: If tumors are radioresistant what, if any, value is there in preoperative and postoperative radium therapy?

I think that this is a most encouraging presentation and that one may well feel that one is on the verge of a new era when tumors will be individualized properly and results will be obtained that have never been dreamed of.

DR. SAMUEL SALINGER: For many years I have had a deep interest in radium therapy. About 1919, Dr. Otto Freer began to use radium emanation in the hypopharynx and nasopharynx and devised an apparatus that went around the head and held a radium capsule placed in contact with the lesions. I watched with him numerous cases such as those Dr. Cutler described which, despite early success, later gave us much disappointment. First he used divided doses; when he found the tumor cells resistant he used larger doses, and then terrific reactions were produced. Finally he felt that if he could devise a technic that would deliver a large dose within a minimum time, without danger to the normal cells, that would be the solution of the problem. But such a technic was never evolved because of difficulties too numerous to mention. Then came the use of bare radium seeds, and with this method we saw lesions in the base of the tongue and the tonsil heal and remain well for two, three or four years, after which the patients had metastases elsewhere. For this reason and also because of a terrific amount of sloughing we were forced to abandon the use of bare seeds.

Then came the gold implants and the platinum implants, and while they were a little more successful than the plain glass tubes we found that we could not accurately irradiate the tumor, for we did not know how far it extended, and the distribution was generally inaccurate. So after several years we gave up the treatment of carcinoma of the tonsil, the hypopharynx and the pharynx with radium, until we began to hear of the radium pack. One talked of giving 200,000 and 300,000 milligram hours, tremendous doses compared to those of our previous experiences, without local reaction.

A case that is in a more or less experimental stage is one of a carcinoma of the anterior commissure, growing up toward the epiglottis. The patient is a man who is not in a physical condition to withstand laryngectomy; so we decided on this treatment. He has received 300,000 milligram hours without edema in the larynx. It is almost inconceivable that the tissues have been able to endure such an enormous dose without damage to the surface and without edema. The lesion is beginning to disappear, and he has marked hyperemia of the mucosa, which Dr. Cutler mentioned, approaching the exfoliation he hopes to secure. I feel much encouraged as to carrying on experiments of this nature. There are many carcinomas of the tongue, the hypopharynx and the tonsil that are absolutely inoperable. One can only add to the patient's misery by operation, and if the method described by Dr. Cutler can kill the carcinoma cells without harming the surrounding tissue, it marks a decided advance in therapy.

DR. ROBERT SONNENSCHEIN: I am not in a position to add anything to what has been said, but I hope that Dr. Pearlman will give the details of the case in which Dr. Cutler has been treating an enormous tumor which within a short time has melted almost to nothing.

I hope it will not be considered in bad taste or immodest for me to say a word about the organization of the tumor clinic at Michael Reese Hospital. My associates and I are fortunate in having there 4 Gm. of radium, than which there is no larger amount in the United States except in the Memorial Hospital in New York. But we are even more fortunate in having on our staff Dr. Max Cutler, who has been associated with the Memorial Hospital Clinic, and we hope that this will be an advantage to the entire territory surrounding Chicago as well as to Chicago itself. We trust that all the members of this society will avail themselves of the facilities afforded there.

DR. A. A. HAYDEN: I wish that Dr. Cutler would explain why he uses the word "sterilize" in connection with cancer cells.

DR. HOWARD C. BALLENGER: I have just performed a necropsy on a carcinoma of the epipharynx with direct extension to the base of the brain. The patient was first seen in 1927. At that time he had metastasis in the glands of the neck, and he also had the typical facial syndrome, including pain in the face. He was treated by radium and roentgen radiation. Except for the last six months of his life this man lived for the five years from 1927 to 1932 in comparative comfort. He went to business every day and attended social functions; he was very comfortable under radium treatment and roentgenotherapy, but he finally died in January, 1932. Two interesting changes were found at necropsy: total destruction of the second nerve and total destruction of the eighth nerve. So far as I know, the eighth nerve has never been reported as being destroyed by carcinoma of the epipharynx affecting the base of the brain. Dr. George Suker told me that involvement of the second nerve from a similar cause is equally rare. This case will be reported in detail at a later date.

DR. M. REESE GUTTMAN: Dr. Cutler has been the source of inspiration in my attempt to study the histologic structure of tumors and to evaluate their microscopic picture and correlate it with the biologic properties of the disease, as well as with the results of various types of therapy. At the University of Illinois, Dr. Lederer and I are attempting work along these lines, and we are especially interested in the results that surgical treatment and irradiation produce in the various histologic types of tumors. Recently I reviewed thirty-seven cases of

laryngeal carcinoma in which operation was performed by Dr. Beck and me, within the past five years, and classified their histologic structure. Thirty-five of these malignant growths were of the adult, more or less fully differentiated type of squamous cell epithelioma. Two proved to be transitional cell carcinomas. We were unable to find a single instance of lympho-epithelioma in these thirty-seven cases. From the results in this small group it might be said that as a rule most tumors of the larynx are not radiosensitive.

In a similar study of seventeen tumors of the nasopharynx, six, or practically one third of all the tumors, were radiosensitive, four of them being transitional cell carcinomas and two lympho-epitheliomas. This high percentage of anaplastic cellular growths found in the nasopharynx can well account for the fact that many of these tumors have been misdiagnosed and miscalled sarcoma. Four so-called round cell or mixed cell sarcomas of the nasopharynx that occurred in adults proved on recent reexamination to be lympho-epitheliomas. A review of six cases of tumor of the base of the tongue shows that four growths were radiosensitive lympho-epitheliomas or transitional cell carcinomas. From this small group of malignant growths about the head and neck it is seen that an exceedingly large percentage are radiosensitive and offer an excellent field for irradiation therapy.

The use of the radium bomb or pack alone, without the addition of interstitial irradiation by means of needles or seeds is certainly adequate to care for the highly radiosensitive type of neoplasms that have been discussed at this meeting. As is well known, they require relatively small doses of irradiation to cause their disappearance, some responding even to such small doses as two and one-half erythema doses distributed throughout the lesion. However, the more adult, fully differentiated type of squamous cell epithelioma requires much larger doses of irradiation, some even more than twelve or fourteen erythema doses distributed throughout the lesion. In several recent communications emanating from Dr. Douglas Quick at the Memorial Institute in New York, he has made the statement that with present technical methods not more than two and one-half or three skin erythema doses of irradiation can be delivered in the midline of the neck and nasopharynx without causing extensive destruction of the skin. If this be so, it is certain that added irradiation delivered by interstitial methods with either seeds or needles must be depended on when one attacks the more adult, fully differentiated type of squamous cell carcinoma. It might also be well to bear in mind that the term radiosensitive does not imply incurability. In fact, radiosensitivity is a characteristic found most frequently in highly cellular, anaplastic, early and widely metastasizing neoplasms, which are apt to be fatal in a very short time. In fact, many, if not most, of these neoplasms have a wide dissemination of the growth throughout the body before the original primary lesion has gained sufficient size to produce symptoms. The prompt disappearance of the primary growth under irradiation will, of course, have no effect on secondary deposits or on the ultimate death of the patient.

Dr. Joseph C. Beck: I wish to call attention to a table that is applicable to what Dr. Galloway and Dr. Salinger spoke about concerning the treatment of poor people. I have a statement from the Hines Memorial Hospital from January, 1931, to January, 1932, during which time 995 patients with malignant tumors were treated, among which growths there were twenty-six tumors of the larynx alone. At Michael Reese Hospital there is a large amount of radium at hand and Dr. Cutler has charge of the tumor clinic; so there should be something he can tell us from his experience about what is going to happen to all the persons who come to the institution from all over the United States. What can be done in the diagnosis and treatment of these malignant neoplasms? Thus far at the hospital it has been my privilege and purpose to operate or to have patients operated on by members of the department, rather than to subject the patient to indiscriminate or possibly unscientific use of radium, but with Dr. Cutler there I hope that conditions will be entirely changed so that radiosensitive tumors will receive the proper type of radiologic treatment.

DR. MAX CUTLER: I am pleased that Dr. Jaffé agrees with me on the question of lympho-epithelioma. His critical opinion is of great value in this intricate pathologic problem. Dr. Jaffé's explanation of the action of radiation is interesting and agrees entirely with my conceptions of this process.

Dr. Beck raised the question of the radiation treatment of benign tumors. In the Radiumhemmet, at Stockholm, Dr. Berven showed me a group of young patients suffering from nasopharyngeal fibromas who had been treated with roentgen radiation and in some cases with local applications of radium. These patients had been followed for a number of years. The results were remarkable. The principle of this therapy is the use of small repeated doses in an effort to induce restraint rather than destruction of the growth.

Zuppinger's results in the treatment of epithelioma of the tonsil, pharynx and larynx are distinctly inferior to those of Coutard and Berven. Analysis of these reports definitely indicates that a high proportion of the patients in Zuppinger's series suffered from advanced lesions.

In response to Dr. Beck's question as to the use of radium seeds, I am convinced that the use of removable, highly filtered platinum needles containing radium element is superior to the use of gold implants in the treatment of intraoral cancer. There are several disadvantages to the use of gold implants: The filtration is inadequate. A homogeneous, uniform distribution is more difficult by means of seeds than with long platinum needles. The radiation is concentrated in a short focus and is not as evenly distributed as it is in long needles. There is a possible disadvantage in leaving foreign bodies in tissues. All these factors favor the use of removable platinum needles according to the technic employed in the Curie Institute. I have examined many pathologic specimens removed following the insertion of radium seeds, and am impressed with the great difficulties encountered in the attempt to distribute seeds uniformly throughout a tumor. This defect leads to over-radiation of some foci and under-radiation of others. Local recurrences are, therefore, more common following the use of gold implants than after the use of removable radium needles. There are some circumstances under which gold implants are indispensable. The most common condition is in small, localized squamous carcinomas situated far back along the base of the tongue where it is almost impossible to retain long needles in position.

As to the dismantling of the radium "bomb" by the National Radium Commission in London, it is generally agreed that the experience gained from the use of this apparatus in London does not justify a final opinion either for or against this method of treatment.

Dr. Hayden raised the question concerning the use of the word "sterilize." By sterilization of a tumor I mean a complete destruction of the tumor cells.

I hope that I have not overemphasized the value of distance radium therapy, but I am fully convinced both from my own experience and from my observations in other centers that distance radium therapy in the form of a radium pack constitutes our most efficient method of radiation in the treatment of cancer of the tonsil, pharynx and larynx. This position is substantiated by the results of Berven and the experience of Forssell. In the absence of this type of therapy the use of high voltage roentgen radiation according to the Coutard technic constitutes an effective method the results of which should be comparable to those that can be obtained with large quantities of radium.

REPORT OF A CASE: TUMOR OF THE MASTOID. DR. NOAH SCHOOLMAN.

Dr. Schoolman reported a case of a mastoid tumor recently operated on, and presented the temporal bone from a new-born child to demonstrate some of the points that he made.

MOVING PICTURE DEMONSTRATION OF THE MANAGEMENT OF ACUTE MASTOIDITIS. DR. AUSTIN A. HAYDEN.

AMERICAN LARYNGOLOGICAL ASSOCIATION

CHARLES J. IMPERATORI, M.D., *Abstract Editor*

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GEORGE E. SHAMBAUGH, M.D., *President, in the Chair*

PRESIDENT'S ADDRESS: CLINICAL PROBLEMS OF RHINOLOGY. DR. GEORGE E. SHAMBAUGH, Chicago.

This article will be published in full in a later issue of the ARCHIVES.

CHONDROMA OF THE LARYNX: A REPORT OF SIX CASES. DR. FREDERICK A. FIGI, Rochester, Minn.

Knowledge concerning the clinical characteristics of chondroma of the larynx, permitting early recognition and effective treatment, accumulates slowly because of the rarity of these tumors. A review of the symptoms, diagnostic features and treatment, together with case reports of six chondromas of the larynx seen in the Mayo clinic, are presented.

DISCUSSION

DR. WILLIAM B. CHAMBERLIN, Cleveland: I recall tumors that I have mistaken for chondroma. One proved to be a tuberculoma and was dense, hard and fibrous, about as large as a very large hazelnut. The other was a cyst of the larynx.

LARYNGITIS AND TRACHEITIS IN CHILDREN, WITH SPECIAL REFERENCE TO NONDIPHTHERITIC INFECTIONS. DR. T. R. GITTENS, Sioux City, Iowa.

Nondiphtheritic laryngitis and tracheobronchitis simulates true laryngeal diphtheria and is often confused with it. In severe cases, tracheotomy is necessary to relieve serious dyspnea, just as in diphtheria. In the nondiphtheritic type there is a marked tendency for secretions and semisolid masses to form in the trachea and bronchi and produce mechanical obstruction of a serious nature for days or even weeks after tracheotomy. Suction through the cannula, together with instillation of weak epinephrine or saline solution to liquefy secretions, is of great importance in the relief of posttracheotomy dyspnea. The giving of fluids freely by mouth, rectum or intravenously is the most important part of the general treatment. Tracheotomy is preferred rather than intubation, for various reasons given in the paper.

Twenty-four personal cases of nondiphtheritic laryngitis and tracheobronchitis were discussed.

DISCUSSION

DR. CHEVALIER JACKSON, Philadelphia: For many years we have been observing a number of these cases, and the outstanding feature of the disease is the fact that it is not recognized, as it is wrongly recorded in the literature. It has to be searched for under the head of membranous and not nonmembranous croup. Pseudomembranous croup is another name for it. It has to be searched for in the older literature under the name of catarrhal fever.

The question of the thymus gland has been brought up. Undoubtedly, the thymus is blamed for many things that do not concern it, but perhaps few cases have been mislabeled thymus disease. Much more often they are called pneumonia. A child dies of pneumonia. The dyspnea, or whatever there may be of it, is thought to be a pneumonic dyspnea. That child is dying of bronchial obstruction, and over and over again children die of a condition diagnosed pneumonia. There is not any pneumonia; there are an atelectasis and an obstruction

of the bronchi, and unless tracheotomy is done and the lower lobes are drained, the child will die.

Diagnosis is always difficult. I have rarely felt that I was justified in withholding the use of antitoxin. There are certain cases of diphtheria in which the membrane appears late and in which the Klebs-Löffler bacillus cannot be found, and yet there is real diphtheria, so that I often have sanctioned the use of antitoxin when the disease was eventually proved not to have been diphtheria.

The chief means of diagnosis is, I think, direct laryngoscopy. When the child is croupy, the cause of the croupy cough should be determined by looking at the region that causes croupiness in cough; in other words, the larynx and, especially, the subglottic tissues, which swell in diphtheria, in this disease and in a good many other diseases, should be examined.

On the whole, I agree with Dr. Gittens that tracheotomy in this disease, a low and properly done tracheotomy, is preferable to an intubation.

Under that treatment almost all patients will recover, and toxemia is not so much a factor as the lack of good plumbing.

Dr. Mervin C. Myerson, Brooklyn: This is an interesting clinical entity that Dr. Gittens has brought to our attention. There is, however, a group of cases which he has not mentioned and which are important from the standpoint of diagnosis, but not so important from the standpoint of treatment, because it appears that nothing can be done for the patients: the acute fulminating type of the same infection, which seems to end fatally uniformly. We see, on an average, two or three of these cases each year, and the history and outcome are about the same. The condition usually occurs in infants from a few weeks to about 18 months of age, and the history is that the baby was left on the floor in the presence of another child, and suddenly turned blue. There is urgent dyspnea, and the parent becomes alarmed and takes the child to a physician to find out what is wrong. The physician, usually because of the high temperature and the dyspnea, rushes the baby to the hospital. Within a few hours after the onset of the acute dyspnea, the infants present evidence of severe generalized involvement of the lungs.

On medical examination, the general practitioner or the pediatrician believes, because of these symptoms, that there is a foreign body involvement. In the first two or three cases of this kind which I encountered a few years ago, I performed bronchoscopy and found nothing.

A majority of these cases fortunately have been autopsied. The organism that is the offending one is Streptococcus hemolyticus. It appears to be a true pleural, bronchopneumonic type of infection, and it appears that we cannot do much for these unfortunate infants. They die within a few hours after admission, and it seems that the duration of the disease is less than twenty-four hours.

Dr. Clement F. Theisen, Albany, N. Y.: Twenty-five or thirty years ago, when Dr. Jackson was starting his wonderful work that has revolutionized the direct investigation of the air passages, I performed one of my first tracheotomies on a child, and I am sure it was for a case of this sort. But there is one feature that occurs to me, and that is that the difficulty in breathing is not always due to the tenacious membrane, but to localized or generalized edemas.

You probably remember the work of Welch, which was done a good many years ago in the department of pathology in Johns Hopkins Hospital. He found that in streptococcic infections of the pelvic organs there is a localized and generalized edema. The same thing happens in the upper air passages, particularly in the type of infections caused by varieties of the streptococcus. There is a localized or a more generalized edema, and difficulty in breathing is caused not so much by the tenacious membrane in the trachea or bronchi, as by the localized edema.

Intubation does not do much good. You have all had the experience that immediately after passing the intubation tube you had to withdraw it at once, because if edema was present, the intubation tube did not do any good, and if there was much tenacious membrane, the tube was plugged up immediately. So I think that Dr. Myerson is quite right that intubation should be tried first, but

personally, in such cases, I have always performed tracheotomy and the subsequent treatment that Dr. Gittens recommended.

DR. EDWIN MCGINNIS, Chicago: I left a case such as that Dr. Gittens described in Chicago. This case was interesting because in the history there was a possibility of the inspiration of a peanut, and one of the younger physicians in Chicago was doing a bronchoscopy — in fact, he had done two bronchoscopies —and his observations, when he turned the case over to me, were that he had difficulty in finding the opening in the right main bronchus. The left was perfectly free. This was proved to be so.

This youngster was about 2 years old. He had difficulty in breathing, which was not laryngeal because he had a pretty clear voice, and the resident said that that ought to be a differentiation between laryngeal edema and subglottic swelling. A few pieces of peanut were found in the right main bronchus and its membrane. Cultures were grown.

The child did fairly well for twenty-four hours, and then he had a recurrence of the difficulty in breathing. I might add that before we performed a bronchoscopy the left lung was absolutely clear. The right lung had patch areas where breath sounds could not be heard at all, the thing you talked about, Dr. Jackson, probably in the bronchi.

We received a call within twenty-four hours to come to the hospital, that the youngster was having difficulty. We thought that it would be best to do a low tracheotomy on him, and that is what we did. The trachea was full of thick, tenacious secretion. Quickly thereafter the youngster commenced to pick up, and a retraction that was present before the tracheotomy immediately subsided.

We instituted the after-treatment that Dr. Gittens enumerated: an oxygen tent, fluids and aspiration through the tracheal tube, and I hope that the youngster will be alive when I return to Chicago.

DR. HARRIS P. MOSHER, Boston: I had a surgical awakening about ten or fifteen years ago. That surgical awakening was in connection with a nondiphtheritic infection of the trachea and bronchi, the infection being caused by Staphylococcus albus or other organisms that generally invade that region. These organisms cause a thickened membrane in the trachea and bronchi. Such cases have been recognized in the contagious department of the Boston City Hospital by Dr. Blades for at least ten or fifteen years. They are desperate cases. A few of the patients can be treated through the cords, but most of them come to tracheotomy. The treatment outlined by Dr. Gittens should be followed, but it is not always simple or satisfactory. The patients who have recovered have gotten well by suction applied through the tracheotomy tube.

DR. GORDON BERRY, Worcester, Mass.: I wish to ask Dr. Gittens whether the opening of the tracheotomy wound in these unusual infections has ever given an opportunity for infection of this membranous type to lodge on that wound.

I should like to register in favor of the use of antitoxin in nondiphtheritic cases.

DR. WILLIAM B. CHAMBERLIN, Cleveland: I should like to ask Dr. Gittens how much difficulty he has had in decannulation in these cases.

DR. CHARLES T. PORTER, Boston: We have had a number of these cases, and in the main have followed the treatment outlined by Dr. Gittens.

I think that the cases can be divided into three classes: The mild class does not require any more than the croup tent, steam inhalation and careful observation, and a great many patients get well without any operative procedure. Another class of cases is the type in which abundant and tenacious secretion exists, which requires constant attention on the part of the nurse and the physician, not only with the bronchoscope, but to clean out the secretions almost hourly. It is almost impossible to get it out with suction. Another class of cases is the type with a dry, scaly, scabby secretion that has to be removed by direct bronchoscopy through the tube and by picking out the scales.

The length of life in the fatal cases that I have seen has been about five days. In a great many such cases, particularly the ones in which there is a staphylococcic infection, at autopsy not only an edema, but a marked thickening of the lining of the small bronchi and the secondary bronchi, which completely shuts off the lung beyond that region, has been shown at autopsy.

The treatment in these cases, either surgically or with constant suction or drainage, would amount to the same in the long run, because such cases are always fatal, and there is no possibility of shrinking the thickened lining membrane and getting any result.

I agree with Dr. Gittens in mentioning intubation only as a life-saving, temporary expedient; also, the tracheotomy must be performed low.

DR. T. R. GITTENS: Answering Dr. Jackson first in relation to the literature, I made a hurried comment, but the bibliography includes about thirty-five references, and the cases that have been reported number anywhere from one to twenty-four, so it is not a rare condition, even as it is reported in the literature.

Dr. Jackson mentioned the thymus. It should not be confusing, but the main point I wanted to make was that often when the child is having rather serious dyspnea, too much time is taken in taking roentgenograms of the thymus and in thinking about it, when there is no use discussing it at all at that time. If the dyspnea is serious, it does not make much difference what it is coming from; we should relieve it first.

Again, Dr. Jackson, concerning pneumonia, one of the main points that I wanted to bring out was that constantly, all over the country, in the hands of pediatricians and general practitioners a great number of these cases are undoubtedly handled as bronchial pneumonia without any thought being given to the condition being an obstruction of the air passages with an atelectasis.

We give antitoxin in all of these cases, because there is no way to be sure with what we are dealing, and we have never stopped giving the antitoxin just because we felt that the conditions were nondiphtheritic, because you cannot be sure, as Dr. Jackson mentioned. One finds Klebs-Löffler bacilli in some cases and not in others.

Dr. Myerson raised a question as to the fulminating type, the acute cases. As bacteriologists mention a good many organisms that may be the cause of this condition, I cannot decide to which one it is due.

In answer to Dr. Theisen's question, it is usually, I should say, a subglottic swelling that causes the main obstruction. There may be no membrane and no exudate, and often the cords are not badly swollen, but in the same case with marked dyspnea, there will be a subglottic swelling, and there may be a moderate swelling in the trachea, so that most of the obstruction is possibly from the subglottic swelling.

Dr. McGinnis brought up the subject of foreign bodies. There were three cases in our series that were associated with foreign bodies, possibly the inhalation of peanuts, and naturally in work on the foreign body, especially peanuts, there will be cases of this type. We will have the same problems, three of which I described. After removing some particles of peanuts, we had the same problem of secretions collecting below the tube and having to be removed. The pathologic process was just the same, and the complications were just the same with the foreign bodies as with other cases.

Of course, the thick secretion in the trachea at the time of tracheotomy has been almost universal in the cases we have seen. The dry stage comes after tracheotomy.

Dr. Mosher mentioned the interesting group of cases caused by Staphylococcus albus. In 1924, Dr. Lynch reported five cases within a month of heavy membranous formation in the larynx and trachea due to Staphylococcus albus, and he had a long, hard siege. He did not decannulate any of the patients for four or five weeks, but they all lived.

Dr. Berry mentioned the possibility of infection around the wound. That is something we have not seen happen.

In regard to Dr. Chamberlin's question about decannulation and his reasons for not using it in the reports in the literature nearly every one else has had much more trouble decannulating than we have. Our average has been eleven days.

THE TREATMENT OF CICATRICIAL STENOSIS OF THE LARYNX, WITH SPECIAL REFERENCE TO THE APPLICATION OF SURGICAL DIATHERMY. DR. M. F. ARBUCKLE, St. Louis.

A brief consideration of the methods now employed in the treatment of cicatricial stenosis is presented, with a review of the recent literature on the subject and a further report on the use of skin graft to prevent the recurrence of scar tissue in the larynx.

Dr. Arbuckle emphasized the necessity for careful and repeated observation of the larynx during quiet respiration in order to gain more accurate information of the distribution of scar tissue and of the possible interference with or departure from normal motility of the larynx.

The plan followed in applying surgical diathermy for the removal of scar tissue from the larynx is described, with a comparison of the results obtained by this and other procedures.

Anesthesia is, of course, necessary; chloroform is used when diathermy is to be employed.

Notes regarding special preoperative preparation of the patient and postoperative care, including indications of damage by chloroform, will be discussed.

The use of carbon dioxide and oxygen in all postoperative cases as a means of prevention of pulmonary complications is discussed.

DISCUSSION

DR. CHEVALIER JACKSON, Philadelphia: Like all surgical measures in laryngeal stenosis, diathermy must be considered from the point of view of two separate and distinct classes of cases.

In the first class there are the adults. You can go as far as you like with surgical measures in adults. In the second class there are the children. In children, I think that conservatism is called for, because you can get something in them that you cannot get in an adult. I have frequently alluded to this factor, but one reason or another has caused it to be overlooked, and no one seems to pay attention to my allusions or, possibly, illusions. The factor in children is growth of the larynx. If you do not traumatize the larynx and you force the larynx to grow, as the child grows the larynx will grow larger, and as the larynx grows larger the lumen will grow larger.

This is a different thing from the old-fashioned statement by the family physician, "Let him alone, and he will outgrow it." He will not outgrow the condition unless you force growth through the larynx; force the function of the larynx by forcing the child to breathe through the larynx, and then the larynx will grow.

How are you going to force the child to breathe through the larynx? By the use of cork, partial corking or skilful corking, allowing a bypass of air just sufficient to induce the child to indraw a little at the suprasternal notch. If you force him to breathe through his larynx to an extent that is possible, without working any injury to the child, you will force the larynx to grow.

DR. J. B. GREENE, Asheville, N. C.: There seems to be a general idea that you cannot use diathermy while you are giving ether. I think that this is rather a misconception. The anesthesia from ether is produced by the absorption of the ether by the blood stream. It is not produced by the amount of ether in the bronchi. If one allows the patient to take two or three breaths of air, the bronchi are quickly freed of that ether. There is no danger of the ignition of the ether by the discharge of the spark.

I do a good deal of work with diathermy in the larynx. I do it under local anesthesia, however, and seldom under general anesthesia. The work under local anesthesia is easy, just as simple as it is under general anesthesia, and in cases of cicatricial stenosis the application may be made frequently and with little discomfort to the patient.

The last patient with stenosis of the larynx whom I treated was an adult. The case was one of subglottic stenosis, which occurred twenty years after a probable ulceration contracted during typhoid. The patient had a very small opening and had a great deal of difficulty in breathing.

I think that in adults, especially in those pretty well along in age, the use of dilators is successful and is to be preferred to various operative procedures that have been described in the literature.

Dr. M. F. Arbuckle: I am tempted to ask Dr. Jackson a question, which he can answer now or later as he sees fit: Does laryngofissure in children arrest the development of the larynx?

In regard to Dr. Greene's comment as to the safety of applying diathermy in the presence of ether anesthesia, I have not the courage to do it.

I believe that chloroform is a good anesthetic, and that if it is properly given it is a safe anesthetic.

Regarding the use of diathermy or any other form of treatment except dilatation, with local anesthesia or without anesthesia in children, I think that the children in California must behave better than those in Missouri, because I am not able to use diathermy or any other treatment that requires great accuracy in the larynx with a local anesthetic.

Presentation of Instruments and Specimens. Dr. Thomas E. Carmody, Denver.

Osteomas in the sinuses are rare.

A man, aged 26, a medical student, had been seen by a number of nose and throat men and once by our President, Dr. Shambaugh, with a severe headache. The headache was relieved after two or three days as a rule, because drainage occurred from the sinus, but roentgenograms were not taken until after he had had severe headaches for almost four years, and he had had them almost continuously for the last four months. The osteomas were both in the left frontal sinus. They interfered with the drainage sufficiently so that the symptoms were produced.

In this case an ordinary Lynch operation was done and the specimen removed, and entire freedom was obtained. However, there is one peculiar thing about this case. In the opposite sinus, the right frontal, there was an apparent polyp. The polyp in the other sinus remained, and there was a possibility that there was a potential osteoma in that sinus.

The second patient was a man, aged 37, a war veteran, who gave a history of the development of emphysema of both of the eyelids on the right side three years previously after blowing his nose. There was apparently no examination at that time. On Jan. 1, 1932, he developed an aphasia while talking business, and was unable to proceed with his business arrangements, but he recovered his voice on returning home that evening. The next morning he found that he was paralyzed on the right side, the arm and leg; the paralysis remained for some days, when he began to recover the use of the right arm. The leg had not recovered or had recovered very little at the time I saw him on April 14.

A Killian operation was performed on April 17, and an attempt to do the ordinary operation, like the Lynch, resulted in inability to remove the specimen. The specimen was attached to the posterior wall of the frontal sinus, and when it was pulled away there was a hissing sound. Apparently quite an amount of air or gas and some fluid were present. There was an opening through the dura, the dura was necrotic, and there was apparently an abscess of the brain. The lateral roentgenogram showed the air extending back over the parietal region.

Recovery was uneventful, and the patient was recovering somewhat the use of his leg. After leaving the hospital, he was placed under my care in the Veterans' Bureau Hospital; I saw him on the day I left Denver, and he was in good physical condition.

DISCUSSION

DR. LEE WALLACE DEAN, St. Louis: Why did you say that the second patient probably had an abscess of the brain?

DR. THOMAS E. CARMODY: In answer to Dr. Dean's question, it was questionable in our minds because of the lack of a laboratory report. That is, we could not say absolutely that there was pus from the anterior part of the brain cavity, but we felt that there was certainly an abscess of the brain, because there was considerable infection in the frontal sinus; we thought that this was an abscessed cavity which was partially filled with gas or air. If you will notice the x-ray picture afterward, you will see that it shows no evidence of air in the brain cage, while the one taken before removal of the osteoma shows evidence of air extending back to about this region.

DR. MERVIN C. MYERSON, Brooklyn: I have a cautery handle I should like to show. It was designed because all others I have encountered have the connecting cord separated; they slip on to metal binding posts, and they become loose and fall off the projecting metal posts, occasionally giving the operator a shock. Also, there is usually some arrangement with which you make contact, which gets out of order readily, and the arrangement for securing the cautery electrode is such that there must be an insulation in the arrangement, which seems to get out of order.

One great advantage is the fact that any electrode can be introduced into the tubular portion of the long piece of metal in the handle. This can be held firmly and will not go up and down loosely while the cautery is being used.

PAPILLARY CARCINOMA OF THE MAXILLARY SINUS. DR. JOSEPH H. BRYAN, Washington, D. C.

A patient had been treated for several months for an abscess in the left maxillary sinus. Frequent punctures had been made without relief. The patient suffered from great pain over the sinus, which radiated to the frontal region, and frequent hemorrhages from the left side of the nose. Roentgen examination showed marked opacity over the left maxillary sinus; all the other sinuses were clear. A radical operation was performed on October 8, and revealed the left maxillary cavity filled with a papillomatous growth. The operation was attended by severe bleeding. After-treatment was given with radium needles, and there was no return of symptoms after seven months.

DISCUSSION

DR. THOMAS E. CARMODY, Denver: I was very much interested in this report, but I should like to speak about recurrences. Dr. Bryan speaks of no recurrence in seven or eight months.

I recently reported three cases of carcinoma of the maxillary sinus, although only one of them was primary; recurrences took place seven, nine and ten years after operation. The patients had been treated with the Percy cautery or the electric desiccator, and radium and x-rays had been applied. The three patients, I am glad to say, have been cured, and it is now twelve, ten and about eight years, respectively, since the original operation.

However, the point I wish to make is that the old rule that five years after operation you can consider the patient as cured, cannot be taken as true any more, because in the three cases mentioned the shortest time before recurrence was about seven years, and the longest time, ten years.

DR. E. R. SPENCER, Boulder, Colo.: There is one interesting feature that Dr. Greene, of Boston, brought out a few years ago in connection with malignant conditions of the antrum, and that is the importance of having a large opening through the canine fossa, so that the progress can be watched over a period of years. I believe that most malignant conditions in the maxillary sinus are usually not so highly malignant. Dr. Figi, will you correct me if I am wrong?

I believe that Dr. Figi's statistics, as well as Dr. New's, bear me out in that statement, but if a large window can be left so the process can be watched, a recurrence can be taken care of early, before an extensive recurrence or metastasis occurs.

DR. J. H. BRYAN: I just want to caution everybody that this is just a preliminary report, and, of course, if anything does recur, it will be noted.

A REVIEW OF SINUS-CHEST INFECTIONS. DR. WILLIAM V. MULLIN, Cleveland.

It is contended that infected nasal sinuses can and do produce enlargement of the bronchial, peribronchial and mediastinal glands. This, in turn, will produce cough, which, if prolonged, will cause dilatation of the bronchi with retention of secretion. In about 20 per cent of patients with chronic sinus infection, cough is a prominent symptom.

The experimental evidence previously presented has been widely misunderstood and misinterpreted and has probably led to many unnecessary operations on the sinuses.

DISCUSSION

DR. R. A. FENTON, Portland, Ore.: It is cheering to have Dr. Mullin's warning against indiscriminate operations on the sinuses. We have had at times somewhat of an epidemic of that sort of thing along the west coast among some of our more radical brethren, and I think that this warning and the revival of what has come to be an authoritative statement in the literature by Dr. Mullin are something for which we should all be thankful.

In regard to the matter of direct swallowing, the inhalation method that Dr. Mullin pointed out by inks and iodized oil, I think it might be considered that accumulations of rather thickened purulent material in the large bronchi or at the bifurcation will irritate the special nerve endings, which were discovered by Larcel, the things we are always running into when we put a bronchoscope into that region, and that might also be a trigger for the sort of thing Dr. Mullin pointed out in respect to the enlargement of the glands.

It should be a matter of pride to us, also, that this work of Dr. Mullin's, although he was not chasing histiocytes at the time, anticipates by more than ten years the work of Jannuzzi and the other Italians who were pioneers in investigating the reticulo-endothelial system of the tracheobronchial tree, larynx and tonsils. This was the first title I have been able to find in the literature of anything of this kind, and Dr. Mullin's work antedates it by ten years. It is true, in quoting his old work, that he refers to what are now called histiocytes and what Jannuzzi called the reticulo-endothelial system of the tracheobronchial tree, larynx and tonsils as endothelial cells.

DR. F. R. SPENCER, Boulder, Colo.: In Colorado we see patients whose conditions are diagnosed pulmonary tuberculosis when they are not tuberculous at all. The patients have a nontuberculous infection in the chest from an infected sinus or sinuses. Occasionally, we see patients who have both a tuberculous and a nontuberculous infection. Sometimes we have to be careful when we tell patients that they have nontuberculous infections of the chest, because they fail to hear the word "non" and hear the "tuberculous" part of it, and they think they are worse off than we intended they should.

I believe Dr. Mullin is quite right in saying that this infection takes place not only through the lymphatics but by aspiration. That is in keeping with what we know about many other infections of the respiratory tract. I hope that Fenton's work on the histiocytes and also Anderson Hilding's work on the ciliary movement will do much to help us with nontuberculous infections in the upper and lower respiratory tracts.

DR. EDWIN MCGINNIS, Chicago: One lesson that we get from Dr. Mullin's presentation is that when a group of physicians have a limited practice, they should have a competent chest specialist, so that they will not make the mistake of promising too much by attempting to clean up the nasal sinuses in these cases.

As Dr. Mullin says, when you see a youngster 4 or 5 years of age, you certainly are chary about doing anything radical to the nose or the nasal sinuses. You have all had the experience of seeing chronic conditions of the maxillary sinuses in which pus discharged over the posterior end of the entire turbinate. If you look at the larynx in the mirror, you will see part of it divide; some of it goes into the trachea and, by gravitating, descends into the tracheal bronchial tree. I have seen that occur several times, and the most prominent signs in such cases are the cough and the purulent expectoration.

I think everybody knows that it is hard to produce suppuration of the lungs, an abscess, by implanting bacteria in the tracheobronchial tree.

Dr. Thomas E. Carmody, Denver: I think that this paper straightens out a great many things that some of us probably misunderstood in Dr. Mullin's first paper; however, I think that we should discontinue the use of two terms, that is, radical treatment and conservative treatment. Why not treat these conditions rationally?

In regard to the cough reflex, any of us who have done anything in the shape of bronchoscopy know that the minute we change our bronchoscope from one bronchus to another, or simply move it a slight amount, we cause a cough. Many of us have also had the experience of seeing cases of sinus disease in which cough was produced and in which the cough reflex was entirely abolished after the sinus had been merely washed out.

Dr. E. Ross Faulkner, New York: I think it is twenty years since I was struggling with an attack of tuberculosis and visited all the health resorts in the country, of course coming in contact with numerous others afflicted in the same way. At the end of my period of cure I came back to New York and started to practice my specialty again; I began to realize that a good many of those patients did not have tuberculosis, that a great many of them had an infection in the head. They had the associated cough, and while the cure they had undergone for tuberculosis was probably about the best thing they could have had, the problem was to treat the infection in the head. I began to realize that fairly early as a clinical entity, and it was difficult for us to persuade our lung specialists in New York that such a thing was so. Finally, I think they came to have quite a strong conviction that it was so, and now you will find that the lung specialists in New York are prone to refer patients to specialists for examination of the sinuses.

The Anatomic Relation of the Sphenoid Sinuses to Dorello's Canal: Abducens Paralysis. Dr. Karl Musser Houser, Philadelphia.

This article will be published in full in a later issue of the Archives.

DISCUSSION

Dr. Harris P. Mosher, Boston: These intimate relationships do occur, and I have come to the conclusion that it is not the anatomic relationship or the nearness of the structures that is the deciding cause as to whether or not there are pathologic changes in the majority of cases. The thing in my mind sums up as follows: It would seem as though this must happen, and it almost always does, and that the nearness of the anatomic relationship is the deciding factor.

New Instruments. Dr. Max A. Goldstein, St. Louis.

An artificial larynx, as devised by the laboratories of the American Telephone and Telegraph Company and as frequently used by patients of the late Dr. John Mackenty, was offered, with several improvements and modifications.

A phonograph disk for recording and reproducing speech, including a disk on which a permanent record could be engraved with an electrically activated recording arm equipped with a diamond point, was demonstrated.

A thin aluminum record with a metal lacquer, which was made abroad, was shown; it cost about 2 cents, including the duty. The average aluminum or wax disk records cost from 40 to 75 cents.

The record is engraved by a recording arm, equipped with a diamond point instead of a steel needle. The diamond point is activated by an electromagnet, and the groove made in the metal record is distinctly nonperishable. If the average composition disk is played half a dozen times, the needle wears into the groove and the record becomes imperfect. The disks presented had been played for experimental purposes as much as a hundred times, being left to run all day, and there was no deterioration in the quality and character of the reproduced sound.

The usual artificial larynx outfit was shown. Dr. Goldstein wanted to call attention particularly to the tracheal connections. A lantern slide demonstration of the use of the artificial larynx was given.

DISCUSSION

Dr. D. B. Delavan, New York: This demonstration, in both of its aspects, has been of peculiar interest to me. It is only a few days ago that at the New York Academy of Medicine we had a symposium in honor of the memory of our late member there and here, Dr. MacKenty, and, as you all know, Dr. MacKenty devoted a great deal of time and quite a little capital to the development of an artificial larynx.

At that symposium one of his patients, a gentleman of about 50, who had undergone a laryngectomy, was present, and he demonstrated the use of the artificial larynx. The hall in which he spoke was not a difficult apartment in which to be heard, and its acoustics were very good; nevertheless, it was a hall of considerable size, and yet he made himself thoroughly and well heard through the course of a speech of considerable length. The stenographer who was present took down his comments.

With regard to the phonograph, a number of years ago, when the phonograph first came out, I presented an article before this Association on the prospective value of the phonograph with regard to the recognition of defects of the human voice and of its value to the vocal instructor in aiding him in teaching his pupils.

Dr. Joseph H. Bryan, Washington, D. C.: There is a department in the Library of Congress where these records are kept, and I believe that these records will continue to be deposited there and be of great benefit to future generations.

Dr. William B. Chamberlin, Cleveland: I have a laryngectomized patient, and when I talked to him the first time, I did not realize that his larynx had been removed. In other words, he talks as any one might with chronic laryngitis. You can hear him all over the room. He talks perfectly well over the telephone. He carries on work at his office and talks with his patients.

How he does it, I do not know, and neither does he. There is a friend of mine in Oberlin, Professor Stetson, who has been doing a great deal of work for a great many years on phonetics, so I sent this patient down to see him, and he has made an interesting set of records. He recorded his breathing, the movements of the wall of the chest and the like.

The average laryngectomized patient who talks does so by taking great gulps of air into the stomach, and then he talks as long as there is unaspired air in his stomach on which to act; consequently, he has to stop every so often, and there is a little hiatus every now and then until he refills his stomach with air. The patient mentioned has no phenomenon of that kind. In other words, he talks right along fluently, just as the ordinary person does who has a connection between his larynx and his lungs by way of his trachea.

Dr. H. L. Swain, New Haven, Conn.: Forty years ago, before this Association, Dr. Solis Cohen of Philadelphia presented a case in which in the course of a series of operations he had closed off the trachea permanently from the pharynx, and in which a number of interesting things happened as a result of this method of closure on the installment plan. Among other things, you will remember that hairs grew on the inside of the pharynx, because in turning down the skin some of the hair grew in. The patient could talk so that most of the men in the room here could have heard him from this position, and, sitting there

as I did and hearing, I wondered whether this was purely an accidental occurrence or whether it could be made to happen again. As I had a man with a cancer of the larynx under my charge, I determined to try to see whether he could get a voice in that way. He did. I removed the larynx, and the patient, an untutored German, learned to talk, just as Dr. Chamberlin said, but he had a recurrence in the glands of his neck after that and had to go the way of all the earth for that reason. That patient had no instructions whatsoever given him, except to be told that someone else had done it, and he could, if he tried, learn to speak intelligently so that he could be heard fully as well as Cohen's patient; it was a matter of great comfort to me that he did.

DR. MAX A. GOLDSTEIN: I want to offer my tribute to our late colleague, Dr. MacKenty, for his splendid work in the development of the artificial larynx as it is used today.

The artificial larynx is really a significant piece of mechanism and is important for more than its restrictive use in laryngectomized patients, for we can use it in many instances of various laryngeal paresis. We can use it for ulcerations of tuberculous and syphilitic character, in cases in which the vocal cords have been pretty well destroyed; even in such instances the force of the tracheal air from the lungs is not needed, but it can be obtained directly through the larynx, and the artificial larynx is used merely as a vibrating element in connection with the articulating machinery.

The case that Dr. Chamberlin referred to is one of many. There are two technical reconstructions possible following laryngectomy. One is by the use of the artificial apparatus, as you see it there, and the other is by some form of air swallowing. Some of the patients who come to us at the institute — and we have had something like forty in the last five years — are self-taught; they carry a column of air between the superior and middle constrictors of the pharynx, probably enough to say two or three sentences, and then they have to swallow again and catch another column of air to use with their articulating mechanism. Where the vibrating element comes from has not been definitely determined; probably this peculiar husky speech sound is made in the upper surface of the esophagus, but it is a comparatively easy thing to teach laryngectomized patients the use of this new form of speech mechanics. However, some of our teachers report that it is easier to use the artificial larynx. The time taken with the artificial larynx in the production of fairly good and fluid speech by the average patient is about half the time necessary to acquire speech in this more complicated, but perhaps more natural, way. The speech that you get through the air content of the upper part of the esophagus is not a speech that can be projected, nor is it as resonant as is the speech that can be produced in the artificial larynx.

THE PALE NASAL SEPTUM SYNDROME. DR. D. C. JARVIS, Barre, Vt.

The mucous membrane covering the cartilaginous portion of the nasal septum presents three shades of pallor to the eye of the observer. On the pale septum the clinical pictures of edema are most often built. The pale septum is associated with a characteristic nasal discharge. It also has associated with it a definite syndrome. The pallor of the mucous membrane covering the cartilaginous portion of the nasal septum indicates a definite systemic therapy.

THE TREATMENT OF ALLERGY BASED ON THE CONCEPTION THAT IT IS A POTENTIAL ALKALOSIS. DR. ARTHUR M. ALDEN, St. Louis.

The results obtained from protein desensitization therapy in allergic cases have been disappointing. An explanation was offered of the conception that an alkalotic background, congenital or acquired, is an important factor in allergy. The rationale of the therapy is based on such a conception. Cases were reported.

DISCUSSION ON PAPERS OF DR. JARVIS AND DR. ALDEN

DR. DUNBAR ROY, Atlanta, Ga.: The condition that Dr. Jarvis speaks of in his paper is, of course, a condition that many of us — no doubt, all of us — have recognized, but I have never seen just exactly the pale septum without the paleness continuing over the turbinates and over the mucous membrane.

Even in conditions that we recognize as edematous rhinitis, when we look into the nose we find the whole mucous membrane pale, and yet when we look into the throat, we find a perfectly normal red throat. Therefore, I cannot understand how this condition can be localized just in the nasal cavity. Whether it is due to a local condition entirely, or whether it is a systemic condition, I think is a question that must have further study.

DR. KARL MUSSER HOUSER, Philadelphia: My experience has been parallel to that of Dr. Alden in my relation with the allergist, in that the patient who is referred to him, exceptionally rather than the rule, is improved by his administrations, the exception usually being in the type of seasonal allergic conditions which are due to one or more proteins, in which a vast amount of sensitivity exists as a rule and results are not secured.

I should like to ask Dr. Alden two questions: Have any tests been done to demonstrate whether a hypo-acidity or an actual anacidity of the gastric content exists. Have blood tests been made along with these cases to find out whether or not the hydrogen ion concentration does show an actual shift toward the alkaline side?

DR. MERVIN C. MYERSON, Brooklyn: I should like to mention that it appears to me that an alkaline state of the body seems to favor an increased permeability of the blood vessels. It might be interesting for me to state here that only a few weeks ago, while visiting Dr. Lipman in New York, he took a book from one of his shelves and showed me that a little less than a hundred years ago Kronheim predicted that the understanding of the production of many diseases would be based on the question of permeability of the small blood vessels.

The pale septum in most cases, perhaps not all, indicates an alkaline state of the chemical composition of the body. That is the opposite of our observations of the dark red septum, which indicates the acid side of the chemical reaction of the tissues.

It might be interesting to note that I tested a group of thirty-two patients as they came into my office. The majority showed a nasal septum that indicated a markedly acid reaction. The placement of a piece of red litmus paper against the anterior septum always provoked an alkaline reaction; in other words, the red litmus paper always turned blue, even in a nose that appeared acid. Of course, we know that the normal nasal secretion is alkaline in reaction. That leads to the inference that each patient with a condition of the nose that indicates alkalosis or hyperacidity presents an individual problem as to the chemical composition of the body. We have encountered patients whose septums have indicated an alkalinity, and yet after studying that patient we have found that he had an acid rather than an alkaline reaction.

I might say, also, that when my patients have an extreme picture of the polypi or extreme or excessive moisture of the nasal structures which goes to the pale septum, I do not hesitate to give them as much as 60 minims (3.6 cc.) of nitrohydrochloric acid three times a day.

DR. F. T. HILL, Waterville, Me.: I think that this is an interesting subject and worthy of further follow-up, but I do think that we ought to use a little caution so that we do not get into disrepute. What I have in mind is this: In a patient we had last winter whose symptoms apparently indicated that she should have acid therapy, careful examination by the internist disclosed a gastric ulcer, which would seem to contraindicate the use of medicinal acid by mouth.

The allergists have been criticized as being skin scratchers. I think we all know that we are just scratching the surface of the problem of allergy, and if we are going to scratch deeper along this line and get somewhere, it is going to be a fine thing; however, I do believe that these patients should all be carefully examined, taking all the factors of the body into consideration, and using an internist rather than an allergist to get the whole conception of the case.

I wish I could report as remarkable results as Dr. Alden has done. Of about ten cases in which acid therapy was used, we got symptomatic improvement in one or two, but examination of the nose did not show much change. Probably that is due to our own inefficiency.

DR. THOMAS E. CARMODY, Denver: While both of the essayists mentioned dieting, they did not mention limiting foods. Most of us know that we all eat too much, and I think that in many cases hypo-acidity can be overcome by limiting food—sometimes not by giving acid food, but by restricting food entirely.

DR. JAMES A. BABBITT, Philadelphia: It is not likely that I shall add anything to this discussion. I cannot help, however, paying some tribute to the modesty, patience and absolutely tireless work which my associate from my native state of Vermont has put on this problem, and the admirable report of the study in St. Louis.

After sitting in with Dr. Jarvis at several lunches at French Lick last summer, I became much interested in his theory. I shall not attempt to review the results of application of his principles to about fifty patients until the discussion of the symposium next week.

It seems to me that the one weak point has already been suggested by Dr. Houser's discussion, one which I think was suggested in the discussion in New Orleans, and that is, the indefinite rating of the color index of the mucous membrane of the septum.

It is not at all difficult to determine a very pale septum and a very bright crimson one, but the shadings and modifications which go on in an active irritated nose seem to me rather difficult to handle, and there is as much difficulty as with a hemoglobin index. If some definite physiochemical studies could be conducted, it seems to me that perhaps some index, perhaps photographic, of the variations in color with a consistent checking up by several methods of alkalosis and acidosis might be evolved, and we should have something which would be valuable for follow up. In vasomotor rhinitis conditions, which are so baffling, there are several background factors: the vegetative system (sympathetic and parasympathetic), allergic, endocrine and focal, which might require a check-up before indiscriminate use of either the alkaline or the acid treatment.

It raises a smile in my office when, so frequently, patients are questioned on dietetic habits. Some rather surprising results have followed the corrective advice given. The number of patients who have improved, when it seemed to me wise to give dilute nitrohydrochloric acid in certain measured quantities, has been surprising. There are certain cases in which we give, as a routine measure, a teaspoonful of sodium bicarbonate two or three times a day. I wonder whether Dr. Jarvis has tried in certain of his patients, for routine laxative treatment, the substitution of a fairly large dose of sodium bicarbonate before breakfast. If that were done, I should venture to say that the customary laxative could be omitted.

We have all, occasionally, reached an "impasse" with certain of our patients, worried by the failure of ephedrine, epinephrine and various other treatments for a stuffy nose, which is a positive matter to the patients, even if not positive in our diagnosis. I have been surprised in four or five instances to find that, by simply sweeping the decks clear, omitting everything except a little liquid petrolatum, and applying nitrohydrochloric acid, the field has cleared up.

DR. R. A. FENTON, Portland, Ore.: I have, along with some of the other men, been experimenting on the theories of Dr. Jarvis, and we have had some rather startling results. The thing that we lack in all of this, however, is the accurate laboratory check-up, and I think that that has got to be done by the physiologists and the biochemists for us. We cannot do it; it is not our job. However, these two interesting papers certainly favor conservatism in surgical procedures on the nose, and they remind us, too, that it might not be unwise to read some of the work of the older clinicians, who used to look at the patient instead of at test tubes and through microscopes. We might read Hilton's "Rest and Pain" (New York, G. Bell & Sons, 1892) with a good deal of profit at times

I might suggest an explanation of the color index of the septum, namely, the distribution of the nervus terminalis, which is a part of the sympathetic system that reaches the front of the septum above, but is not connected with the sphenopalatine distribution. There is not much known about the cerebral conditions of that system, but it might be interesting for some of the anatomists to follow the work of Ramadier and Lasazna, who did a good deal of work on pericarotid sympathectomy, to determine the effect of the sympathetic on the mucosa of the septum of the nose and turbinates.

DR. EDWIN MCGINNIS, Chicago: There is another side to the question: that is, it is notorious that country people send all the good parts of their products from their farms and gardens to the city for sale: milk, eggs, butter and various fresh vegetables; they do not eat those products at home, but eat canned goods and very little fruit. You see very few oranges on the farm.

There is another thing to think about in allergy. If you go into the history, you find that the sensitivity was handed down to the patients by their grandfather, mother or father. It is the same as the color of their eyes, their hair and the like. It is pretty hard to change. I got my impetus in allergy years ago, when I first went to Boston and got acquainted with physicians there. A good deal of time was spent in skin testing and in trying to clear up allergic situations, and I went back enthusiastic, but during the years I have found that I have not been able to do much for these patients.

DR. FIELDING OTIS LEWIS, Philadelphia: It seems to me that the explanation of the pale septum is that there is a difference in reactivities. We know that these people manifest in their tissues a difference in reactivities, and that the vascular permeability and the vascular reactivity may be different from what we choose to use in contrast as the normal.

It may be that the vascular reactivity is just as great elsewhere, but at that particular point there is a condensation or a concentration of actual physical traumatization on the part of the inrushing column of air on inspiration, which is sufficient to evoke a visible difference in the amount of blood that is to be seen in that area. However, I have not been able to see it, but it seems to me that that might explain why if it turns out to be true of that area, it should not be true of another area. I cannot imagine that there is a nerve, a sympathetic system or another anatomic reason for it.

If we go through the recent developments with respect to biochemistry of the body, we find a good many interesting and succinct points that are coming out at the places where they best would come out in the laboratory of the biologist, the chemist, the physicist, the zoologist, the physiologist and others with respect, for instance, to the proteins, the study of proteolysis. Investigators have been making great headway in the last five years, greater than has ever been made before with respect to the study of proteins. The workers, Smithberg in Uppsala, Adair in England and others, do not agree as to what proteolysis is. One says that it is a chemical molecule. The other says that it is a mixture, a chemical mixture. Let them find that out. However, the fact that they are on the way is enough for now.

Wright long ago said that the tubercle bacillus is the cause of tuberculosis no more nor no less than the skin is the cause of dermatologies; without the tubercle bacillus there could be no tuberculosis, and without the skin — well, finish that yourself.

I think that that is a wonderful text. I think it is one of the greatest texts that a man can have enunciated, because I believe that we have been overlooking the soil entirely too much.

DR. H. L. SWAIN, New Haven, Conn.: Dr. Jarvis, to what great ills are we to think our patients or ourselves are about to be prone because of pale septums? Is it important that we discover that we are having alkalosis or acidosis?

Those of you who read some time ago an article in the *American Journal of the Medical Sciences* by a nonprofessional man, suggesting, as I remember, the use of hydrochloric acid in hay fever, which seems to be borne out by Dr. Alden's

paper, will remember that the writer said purely empirically that if he gave enough hydrochloric acid to his friends and his relatives he could control their hay fever.

DR. D. C. JARVIS: While one uses the terms "alkalosis" and "acidosis" and speaks of using "acid therapy" and "alkaline therapy," one realizes that those terms cannot be maintained, and that other things happen to explain the results obtained.

Considerable clinical evidence is accumulating which suggests that there is an inability or an ability of a person to burn his dextrose. A patient with a red septum apparently burns his dextrose with difficulty. A patient with a pale septum burns it too rapidly. By means of acid therapy and alkaline therapy, we apparently just regulate the flame. By means of alkaline therapy or alkaline-ash food, we apparently carry the chemistry of digestion a bit further, breaking up the dextrose in the body into finer molecules, so that it burns more readily. Likewise, by giving a little hydrochloric acid or acid-ash food, we hold up this process.

Instead of saying that a patient with a red septum is a potential alkalotic, we should ask ourselves, is that patient a potential diabetic? Clinical evidence is accumulating which leads one to suspect that what is happening is just that.

DR. ARTHUR M. ALDEN: There is a great temptation to generalize and to theorize about this question, but I have done neither. I am afraid to because of my consciousness of my limitations as far as my knowledge of fundamental physiologic chemistry goes. I do not know enough about this thing to theorize, but I have drawn no conclusion. My only conclusions about the whole matter are those as far as they concern my own practice, and that is this, that I have been encouraged by the results I have been fortunate enough to obtain and the results that some of my friends have been able to get with this agent, so I am going to continue to use it in the future with the hope that some day I may find out something about it, a lot more than I know at the present time.

MOVING PICTURE FILM, SHOWING CARDIOSPASM. DR. HARRIS P. MOSHER, Boston.

MUCOSAL IMMUNITY IN THE NOSE AND ACCESSORY SINUSES. DR. RALPH A. FENTON, Portland, Ore.

There has been a recent change in the traditional attitude toward histopathology and immunity. The "fluid matrix" and its dependence on the sympathetic nervous system were discussed. The variables affecting the cell were mentioned. There are regeneration and metaplasia of the epithelium. Connective tissue is the basis of all mucosal vitality. There are phagocytic fixed cells in the nasal mucosa. There is a migration of cells from the blood stream. Immunization is obtained by the action of vaccines on the true skin. Embryonal tissue extracts are of value in fostering growth. There is a necessity for further research in cellular physiology, through diet and metabolic studies. Constitutional factors were discussed. Connective tissue, not epithelium, is responsible for mucosal immunity.

DISCUSSION

DR. GEORGE B. WOOD, Philadelphia: I am much interested in the idea of the fibrous tissue being of especial importance in securing protection against penetration, as I understand it, of organisms in the mucosa, and yet in the careful histologic study we can see that these organisms fail to pass through the epithelial cells unless there is some pathogenicity of the organism.

I was wondering whether it was not more a relationship between the disease-producing powers of the bacteria and what we might call the vital resistance of the tissue, which has to do with the repelling of the diseased organisms, than to any special layer of tissue element that intervenes between the organism and the penetration of the bacteria. It is a vital condition. As yet I do not know about it, and I have not been able to explain or to understand it.

All new ideas in regard to local immunity are, of course, of extreme interest; for instance, in the nasal cavity experimental data have shown that it is almost impossible to infect a normal mucous membrane with bacteria, yet under certain conditions the body resistance would be such that the same organisms would penetrate rapidly and cause disease.

DR. F. R. SPENCER, Boulder, Colo.: A few years ago I had the pleasure of seeing a motion picture by Dr. Florence R. Sabin, of the Rockefeller Institute in New York. The film was not speeded up, but I was impressed by the rapidity with which the macrophagocytes work. Dr. Fenton tells me that they are practically the same as the histiocytes. Dr. Sabin called our attention to the fact that these cells are heavy eaters.

DR. R. A. FENTON: In regard to the penetration of bacteria through the mucosa, I think it is agreed that that is an easy process. If the fluid matrix that contains these protective substances is washed away, bacteria can easily get in, and then, and not until then, does the basement membrane show up. I think Dr. George Shambaugh showed that there was no such thing as a basement membrane, so there was no basement membrane until irritation started. Is not that so, Dr. Mosher?

DR. HARRIS P. MOSHER, Boston: That is partly so. There is always a basement membrane, but it does not have a definite effect until something happens to it.

DR. R. A. FENTON: The fact that in a normal mucous membrane it is not there, or is there in no such defined an area in a membrane that shows irritation, is proof to me that the connective tissue takes up the job of fighting against this infection. Also, there are not very many blood vessels in the epithelium. There is a blood supply such as exists in the fibrin stroma. It is not very thick at first. The more irritation there is, the more blood supply comes in, and the more histiocytes are immobilized.

We commented on the work of Dr. Sabin a couple of years ago when we were first taking up this histiocytus, and at that time we pointed out that there was a considerable difference in the study of this histiocytic defense in the mucous membrane as against that in the serous membranes, namely, in the pleura and the peritoneum. In the one case there is a defined type of epithelium which is fighting against constant infection in the airways, and in the other case there are closed cavities which are immune by reason of their being shut away from the outside world.

THE LARYNGOLOGIC ASPECT OF HODGKIN'S DISEASE: REPORT OF A CASE.

DR. JOHN J. SHEA, Memphis, Tenn.

The difficulties of making a differential diagnosis in cases with enlargement of the cervical lymph nodes are presented in the study of a girl, whose biopsy specimens showed pathologic changes diagnostic of both hyperplastic tuberculosis and Hodgkin's disease in glands taken from the neck and the pharynx: first biopsy specimen, from the right cervical region, Hodgkin's disease; second biopsy specimen, from the right pharyngeal wall, chronic hyperplastic tuberculosis; third biopsy specimen, from the right cervical region, chronic hyperplastic tuberculosis; fourth biopsy specimen, from the left cervical region, Hodgkin's disease; fifth biopsy specimen, from the left cervical region, Hodgkin's disease.

DISCUSSION

DR. WILLIAM V. MULLIN, Cleveland: Dr. Shea's accurate case report brings out several points. One is to pay attention to persistently large cervical glands, and do not attribute them all to chronic infections in the tonsils or the upper respiratory tract. Do not be entirely satisfied with one diagnosis. The third point is the splendid way he has worked out the diagnosis by bringing out the susceptibility of certain animals for the various strains of tuberculosis.

The question arises immediately whether tuberculosis is a negligible factor in this case, and merely one of the childhood infections that we know children become infected with and develop immunity; it stays locked up in the glands and gives them no further trouble. Whether the tuberculosis is of any great moment in this case will remain to be seen by the clinical course.

The susceptibility of these glands to roentgen therapy is another factor.

DR. MERVIN C. MYERSON, Brooklyn: I should like Dr. Shea to tell us what determined the removal of this large mass of glands, and whether he took into consideration the possibility of the glands being tuberculous, because I wish to mention that which we all know, that frequently tuberculous glands erode large blood vessels, so that such a procedure was not entirely devoid of danger.

DR. R. A. FENTON, Portland, Ore.: The value of biopsy in this sort of case is tremendous, and I think that the admirable work of Dr. Shea points a moral and adorns a tale in that respect, and also that of animal culture. In regard to the matter of roentgen therapy, we have made quite a study of that lately in regard to the value or not of irradiation in the treatment of sinus disease. On checking up, it has been definitely shown that these things experimentally destroy the lymphocytes, and the lymphocytes then are picked up by the histiocytes.

It may be true, and probably is true, that the histiocytes have more phagocytic powers in young persons, which might account for the difference in the value of irradiation in young patients as against that in older ones, on whom it does not seem to have such a favorable effect.

DR. JOHN J. SHEA: The pathologic and bacteriologic studies in this case were made by Professor Smyth, of the University of Tennessee, and most of the art work came from that department, the case having been partially examined in their service and partially in our service in a private hospital. The pediatric service, of course, has examined the patient carefully and has studied her chest. I considered her chest negative for tuberculosis because of the roentgen and physical findings.

ANAEROBIC RETROPHARYNGEAL ABSCESS. DR. MERVIN C. MYERSON, Brooklyn.

Anaerobic retropharyngeal abscess constitutes a different entity than the retropharyngeal abscesses that are generally encountered. A discussion of this condition was given, and two cases of retropharyngeal abscess caused by anaerobic organisms were presented.

DISCUSSION

DR. S. IGLAUER, Cincinnati: Dr. Myerson has brought up an interesting subject, and I should like to discuss one feature of it, namely, the presence of air or gas bubbles in the retropharyngeal space. I think he is wrong in assuming that when he gets this picture, which, by the way, is valuable from a diagnostic point in the presence of a foreign body, it necessarily meaps that there is a gas bacillus or an anaerobic infection. It may not be gas at all. Very likely, in a great many cases, as in the case shown (case 2), it is air.

We all know that a patient swallows air at every attempt at deglutition, and that this air is under some pressure. In the presence of a perforated esophageal wall, the air will get into the tissues, so that wherever you suspect a perforation of the esophagus it is important to take a picture, and if you find these bubbles you are not necessarily dealing with a gas bacillus infection, but with a perforation.

DR. MERVIN C. MYERSON: I do not quite agree with Dr. Iglauer about the second patient, because there was a foreign body that acted as a cork in a bottle. You could not possibly squeeze much air, or any air, into those tissues, and if you could you would have a different distribution of the gas. Also, there was a distinctly foul odor on incision in the second case.

THE NASAL ACCESSORY SINUSES AS A FOCI OF INFECTION IN CHRONIC ARTHRITIS. DR. LEE M. HURD, New York.

In the majority of cases of chronic arthritis, there is an involvement in the nasal sinuses (68 per cent), which is usually overlooked because of its latent character. This is brought out in illustrative cases, in half of which the patients had had their tonsils removed; several had had previous operations on the sinuses, without benefit. Only a few complained of a purulent nasal discharge, frequent colds or headaches.

The technic of examination of the sinuses in such cases is important. Careful and repeated inspection of the nose before and after shrinking the mucosa, anteriorly and postnasally, with special attention to the noting of the characteristics of the mucosa of the middle turbinate and the middle meatus, in which changes are rarely absent in involvement of the ethmoids, frontal or antrum, is the first essential. Transillumination is of aid, but may mislead and should be checked by the clinical findings and by carefully made stereoscopic x-ray films, which are of the greatest value. The sinuses most frequently involved in these illustrative cases were the ethmoids and antrums.

The treatment is almost entirely surgical; judgment as to the proper type of operation is difficult. The author often found that he had erred in not being radical enough. A few patients refused operation, and some have shown improvement under medical treatment.

DISCUSSION

DR. R. A. FENTON, Portland, Ore.: I should like to ask Dr. Hurd what the predominating organism was in these cases, or whether bacteriologic studies were made in any of them.

DR. R. A. BARLOW, Rochester, N. Y.: I understood Dr. Hurd to say that a great many of these cases were negative rhinologically. That is perfectly possible, of course. The x-ray films can err just as much as clinical evidence. The cases that Dr. Hurd presented showed positive findings in the antrums as registered on a film by the opacity. That is a certain positive way of determining pathologic changes. I wonder whether Dr. Hurd, in looking over the films, which apparently did not show opacity, and were negative, as it were, would not consider the character of the bony walls, not only in the antrums, but in the ethmoid structure.

There have been cases we have seen in which the x-ray films showed a waterlogged, semifuzzy, semitransparent appearance of the ethmoids. After from six to eight months of treatment, particularly in children, x-ray films again showed a distinct difference in the appearance of those cells.

The condition seems to be of a circulatory nature, and I should appreciate it if Dr. Hurd would look over the old films in these negative cases and see whether he cannot find some change in the bony walls. I think that there is where we must look for hidden evidence not registered as opacity, and if the roentgenologist can get the cooperation of the rhinologist in carefully examining these films, as Dr. Hurd does, stereoptically, I think we shall get somewhere in our future interpretation of these mild inflammatory lesions.

DR. DUNBAR ROY, Atlanta, Ga.: For my own information I should like to know from Dr. Hurd or from some of the other gentlemen here who interpret the x-ray plates for him, whether they make the interpretation themselves, or whether the man who takes the picture makes it.

Dr. Hurd spoke of conservative rhinologic treatment. I should like to have him mention what is conservative rhinologic treatment for patients with cases such as he has presented. That treatment seems to vary considerably; also, it must be considered whether the cases are acute or subacute, or whether they are entirely achronic conditions.

Then I should like to know from the other gentlemen whether they would be perfectly willing to operate on a patient who has to their satisfaction been demonstrated by x-ray plates to be entirely without any decided objective symptoms?

DR. WILLIAM V. MULLIN, Cleveland: In our clinic we believe that there is only one treatment for arthritis, and that is general. That is the method of approach, and we think that it should be used in all cases.

Furthermore, I think that Dr. Hurd's method of approach is just a little bit at variance with this. I object to the term "chronic arthritis." I think that every case of arthritis should be classified before any operative work is done. You can use whatever classification you like. There is a good one put out by the Arthritis Commission, but in a general way it can be classified into hypertrophic arthritis, atrophic arthritis and infectious arthritis.

Two of these classes the hypertrophic and the atrophic are principally of metabolic origin. If after the cases have been thoroughly examined and worked out there is a definite focus that is disturbing the metabolism, then I think it is perfectly right to operate on the patient and remove the focus. However, to start in and remove such a focus before examination is objectionable.

Dr. Dickson, of our clinic, a few weeks ago at our staff meeting reported 192 cases of periarthritis of the shoulder joint. These were not 192 consecutive cases, but cases with complete follow-up records, and every patient who did not reply or report for examination three years after the first time he was seen was not counted. Dr. Dickson showed that there was 98 per cent improvement in the 192 cases, and that the patients were treated in various ways; however, they all did improve, and the improvement occurred between the third and fourth week no matter what sort of treatment was given. I mean that the patients who were operated on and put on a general regimen got their improvement at that time. Some of them obviously had hyperthyroidism and required operation. Others had calcification of the joints to such an extent that they were operated on and the calcification was removed. Therefore, I think that if one approaches these cases from the general standpoint rather than from the removal of one or more foci, one will have a better result.

DR. EDWIN MCGINNIS, Chicago: I had an attack of arthritis and an infected maxillary sinus.

Dr. Mullin is perfectly correct. Some patients have chronic tonsillitis, some have a chronic sinus infection, and some have devitalized teeth. We see such involvement every day in our hospital. Some patients have a chronic condition of the gallbladder and chronic appendicitis, and in some each joint acts as a focus of infection.

DR. LEE M. HURD: In regard to the organisms, Dr. Snyder has his own laboratory, and he says that as long as bacteriologists find twenty different kinds of streptococci, he is not willing to say which organism is doing the damage.

In answer to Dr. Barlow, I operated on a man three days ago who had a pure Streptococcus haemolyticus infection. He had x-ray pictures made two years ago, and a 1 plus ethmoid, sphenoid and antrum were seen. I had an x-ray picture made about a week ago, and his ethmoids looked better, but there were still a 1 plus antrum and sphenoid. We washed out his antrum and sphenoid one or two times, but no pus was obtained. I then operated on him. That night he had a chill and a temperature of 103 F. The condition cleared up.

As to the interpretation of x-ray films, Fineman examines and interprets them; then they are sent to me, and I interpret them; we generally agree.

In regard to classification, Dr. Mullin, they have recently classified these cases as acute rheumatoid arthritis, osteoarthritis or a mixture of the two, and, if you will remember, in the charts a good number of the patients never came near the rhinologist at all. The cases were of the metabolic type.

I have never seen arthritis in a nasty, sloppy, sinus infection. Those sinuses are thoroughly opaque to the x-rays. I have never had a patient who had lesions of the joints.

Conservative treatment is the same treatment that you would give in any chronic condition of the nose. You use nitrate of silver, and in some cases, colloidal packs.

Seven of ten patients had no objective symptoms of sinus trouble.

DR. WILLIAM V. MULLIN: Dr. Osgood was the man I was trying to think of from Boston. In his demonstration he had a series of people who on their own volition were taking an increased carbohydrate diet, and he showed with his barium tests an enormous distention of the intestinal tract. All he did was to take them off their carbohydrate diets and put them on a high vitamin diet; the change was perfectly remarkable; the intestinal walls contracted, and the arthritis improved. This was in arthritic patients.

DR. LEE M. HURD: Snyder was at the meeting in Boston also, and they asked him next year to write a paper on the part of the colon, but, of course, he takes care of the diets. He studies the metabolism. Many of his cases I never see. They are metabolic cases, and they are cleared up without the touching of any foci.

Another thing I have observed is that often after the conditions have been cleared up, the immunity of these patients is so meager that they contract some other condition. One patient, who has been well for five years, is getting a disease of the gallbladder. The patients have to be examined constantly, or they continually pick up other diseases.

HISTOPATHOLOGY OF THE LARYNX ANTEDATING CLINICAL EVIDENCE OF LARYNGEAL TUBERCULOSIS. DR. FRANK R. SPENCER and DR. FREDERICK SUMMERILL, Boulder, Colo.

Recognizing the fact that tuberculous infections of the epiglottis, the base of the tongue and the pharynx produce dysphagia sooner than tuberculosis in other parts of larynx and believing that many patients with pulmonary tuberculosis must have early microscopic tubercles in the larynx long before lesions are clinically demonstrable, the presenters undertook a microscopic study of the larynx, the epiglottis, the base of the tongue and the pharynx at postmortem examinations, specimens of patients who had pulmonary tuberculosis being used.

Each larynx was completely removed at autopsy, with the tongue and pharynx attached. These organs were carefully examined for gross lesions. Small pieces of tissue were cut from the larynx, the epiglottis, the base of the tongue and the pharynx for sectioning.

Abstracts of clinical cases were given, with postmortem observations and the histopathology.

DISCUSSION

DR. FREDERICK SUMMERILL: This work is of some importance to you as clinicians, and I hope that it bears to you a slight degree of what it has brought to me in great proportion as a pathologist, an enlightenment on tuberculous lesions in the larynx.

In one of the six cases presented, in which the patient was treated by electric cautery, for a considerable time it was believed that the lesions of the epiglottis were held, and at the time of performing autopsy the gross appearances were gratifying, but the microscopic observations showed that the lesions were active tuberculous lesions.

The pathologic pictures presented have shown a selected number of tubercles, particularly giant cell tubercles, but on going over all the slides of all the cases, it was not so often that these typical tubercles were observed. The predominant characteristic was an undifferentiated chronic inflammation, with the presence, of course, of lymphocytes and epithelial cells and other chronic inflammatory conditions, and occasionally some leukocytes and typical giant cells. The finding of acid-fast bacilli afforded conclusive proof, and the presence of large nuclear cells was scant. These are sometimes called monocytes, and it has recently been stated, by some investigators that their presence is concomitant with the scant presence of bacilli, yet in most instances in the cases presented the bacilli were scarce, and the monocytes were still more scarce.

Although Dr. Spencer brought to your attention the production of caseation in these lesions, it was comparatively scant, and in the prospective of all these sections there was a tendency to fibrous repair. Of course, when the caseation was on the surface, it was readily given off, and chronic ulceration was the picture that was observed.

As far as the site of the occurrence of these lesions is concerned, there are some interesting points: In all the cases there were no lesions of the trachea up to the undersurface of the true vocal cord, which was the lowest site in one case. The most frequent site was in and about the larynx, in pockets about the cords, in which it is readily conceivable that the organisms can easily be lodged. In the pocket of the vallecula, the lesions were observed high up and not in the deepest part of it.

As Dr. Spencer showed, there was one slide of a lesion with bacilli in the dorsal surface of the epiglottis, but it was only in the epiglottis that they extended lower and not on the surface of the tongue. In several instances the infection about the larynx extended over onto the esophageal surface.

Dr. Lee Wallace Dean, St. Louis: In several of the specimens, Dr. Summerill, you said that the only reason for making a diagnosis of tuberculosis of the larynx was the finding of Bacillus tuberculosis in ordinary inflammatory tissue. Now, where there is tuberculosis of the kidney, taking a case in which the lesion is further away or is infectious about the nose and the ear, we often find the tuberculous organisms present in lesions which are not clinically tuberculous and in lesions which heal as nontuberculous lesions heal. I mean to say that in tuberculous lesions of the body it is common to find Bacillus tuberculosis as a concomitant of ordinary infectious processes. That being true, I wish you would elaborate the statement that you made.

Dr. Frederick Summerill: Answering the question raised by Dr. Dean, it was a picture of chronic inflammatory reactions with the presence of bacilli in the internal organs. The organism is carried, as you say, to the kidney and may cause destructive pictures, which may, at times, or may not be accompanied by the extensive inflammatory reaction. However, there is a picture of the tuberculous condition in these internal organs of soft tissue. The bacilli not only are in the picture of the kidney, the spleen and the liver, but are elsewhere, and it is not always, as Dr. Spencer mentioned, that the presence of the epithelial and giant cells is quite concomitant with the tuberculous picture. We showed you the typical portions and the incidence in which the epiglottis showed the chronic inflammatory picture down the center. The section contained myriads of acid-fast bacilli, and in the same picture in other sections in the series were observable pictures sufficiently typical of the reaction of epithelial cells, and occasionally giant cells had to be considered tuberculous. One cannot take a single section and state that it is absolutely tuberculous. It can be said that it is probably tuberculous without evidence to the organism.

I do not know whether that answers your question fully.

Dr. Lee Wallace Dean: In certain specimens that Dr. Summerill showed, the only evidence of tuberculosis was the presence of the tubercle bacillus. There were no cells shown that were characteristic of a tuberculous lesion. All of the cells could easily have been inflammatory cells, which would be present in nontuberculous lesions. When there is an inflammatory lesion, it does not necessarily mean that it is tuberculous. It is common for these organisms to be present as concomitants. If a patient had tuberculosis in some part of the body and inflammation in another part of the body, that might be the picture. That is the point I want to clear up, how you know that the presence of the organism in itself indicates that the condition is tuberculosis.

Dr. Robert Sonnenschein, Chicago: It has been shown by Dr. Spencer that microscopic lesions are often present before demonstrable objective signs can be had. It certainly behooves us in all cases of tuberculosis to watch the larynx most carefully all the time, even though definite signs or symptoms may still be

absent, and to look for the possible lesions that may occur in the lesion which we all know as miliary tuberculosis, the so-called tuberculoma, subglottic edema and the like.

DR. WILLIAM V. MULLIN, Cleveland: Dr. Allen Krause has shown that in the spread of the tubercles throughout the body, they pass through a certain point many times for the one time that it is infected with tuberculosis. He also has shown that in the allergic animal, the spread of the tubercle will make a complete circuit of the body against the lymph stream and the blood stream, say in a period —I do not know that I have his figures exactly right—in approximately four and one-half hours, against eight hours in an immune animal.

DR. J. B. GREENE, Asheville, N. C.: Cases of inactive tuberculosis of the larynx in which there are pulmonary lesions interest me. I should suggest diathermy; the patients should have a vocal rest, as should all those with pulmonary conditions, to avoid a possible lesion in the larynx. There was one other thing that was suggested by this paper, and that is the use of the cautery. The lesions show perfect healing after the use of the cautery, and this emphasizes it as a valuable therapeutic measure. In spite of the pulmonary death of one patient, the lesion of the epiglottis showed perfect healing.

DR. FRANK R. SPENCER: I have talked to Dr. Summerill's chief, Dr. Johnson, professor of pathology, as well, and both assure me that where there is the typical microscopic appearances of tuberculosis, such as giant cells, epithelial cells and lymphocytes, which are there because of inflammation, and then the organisms are found to be caseous acid-fast bacilli, that is as nearly complete as modern medical science can go. In other words, in the eighteen slides shown, the acid-fast bacilli were all present in typical microscopic lesions of tuberculosis.

Sir James Dundas Grant, of London, has been one of the strongest advocates of rest of the larynx in tuberculosis. Some of you who are older in this society will remember the paper on the prognosis of laryngeal tuberculosis that he read here. He made rather startling statements, and he brought over a patient who did not speak a single word, not even by whisper, for one year; the second year he whispered. This patient certainly had a complete cure of his larynx, and Grant's faith in that method of treatment was demonstrated by his willingness to take his own medicine.

THE CAVERNOUS SINUS: ANATOMIC AND CLINICAL CONSIDERATIONS. DR. EDWARD H. CAMPBELL, Philadelphia.

The clinical side of cavernous sinus thrombophlebitis was only briefly discussed, emphasis being placed on the anatomic peculiarities of the sinus itself and the venous drainage of the head and face. The venous connections of the cavernous sinus were reviewed in some detail, and the regulation of the venous blood in the cranium by such factors as gravity, muscular exertion, pressure within and without the cranium and the respiratory cycle was discussed.

In the discussion of the anatomy of the cavernous sinus, emphasis was placed on the narrowing of its lumen by the network of trabeculae traversing it. Consideration was given to the bearing this peculiarity would have on the intracranial circulation and also on a tendency for thrombosis of this sinus. An attempt was made to explain many of the reported cases of thrombophlebitis of the cavernous sinus by a dislodgement of infected particles of thrombus, perhaps close to the original focus of infection, and the transmission of these emboli to the meshwork in the cavernous sinus.

DISCUSSION

DR. MERVIN C. MYERSON, Brooklyn: I was much interested in this extremely instructive presentation. The slowing of the blood current in these sinuses accounts for many cases reported in the literature of the so-called marantic type of sinus thrombosis in the cranial sinus. The cases usually occur in underfed, neurasthenic infants.

It occurs to me that the question of extension of infection by means of an embolus from a vein when the infection is in the lip, as Dr. Campbell said, or when there is an infection of the cavernous sinus from the inferior superior petrosal has another phase; that is, there must be another consideration. That is this: Cases of cavernous sinus involvement of the upper vein as a result of infection, which travels to the cavernous sinus by way of the orbit, I believe have associated with them not only the infective embolic process that Dr. Campbell speaks of, but also an active orbit cellulitis, which helps toward rapid extension toward the cavernous sinus.

The question of approach is important; I think that the intradural approach would be justified every time you feel that there is not a suppurative process inside of that sinus; however, where there is an active suppurative focus, the question of whether or not one might induce meningitis by going in intradurally and opening that sinus, even with diathermy electrode, must be considered. However, if we are dealing with a type of condition that is uniformly fatal, I think that we are justified in trying anything that offers any measure of success.

Dr. William B. Chamberlin, Cleveland: It has been my misfortune to see a number of these cases. I have never seen one following infection in or around the tonsil as Dr. Campbell suggests, nor have I seen any following infection of the mastoid as reported by Dr. Eagleton. One case that I remember distinctly followed the extraction of a tooth. Another case, which I saw in consultation during the past winter, was in a boy 10 years of age following a trivial lesion near the tip of the nose. When I saw the boy he had a high fever and was unconscious; he died within the next ten or twelve hours.

The suggestion of approach by Dr. Myerson is extremely interesting.

Book Reviews

Röntgendiagnostik des Schädels bei Erkrankungen des Auges und Seiner Nachbarorgane. By Dr. Rudolf Thiel, Berlin. Price, 48 marks. Pp. 87, with 415 illustrations. Berlin: Julius Springer, 1932.

This is an admirable atlas on the subject and is worthy of much praise. It shows in roentgenograms a profusion of plates illustrating tumors, infectious disease and systemic disease, which is so located in the skull that it has a direct influence on diagnosis of ocular and orbital conditions whether or not they are primary in the eye or the optic tract. It is greatly enriched by photographs of the patients, schematic drawings to explain the interpretation, anatomic diagrams and, in many cases, a map of the visual fields. The interest naturally centers about the subject of tumor of the brain, orbital tumors and those producing changes about the sella and the chiasm. All of the illustrations are technically to be admired. The technic of the printer is excellent.

The atlas appears in one volume and is accompanied by another volume of text in which the roentgenograms are interpreted.

Plastic Surgery of the Nose, Ear and Face. By Victor Frühwald, M.D. Translated by Geoffrey Morey, London. Price, $4. Pp. 86, with 88 illustrations. Vienna: Wilhelm Maudrich, 1932.

The author presents the subject of plastic surgery of the nose, ear and face in a concise and lucid manner, just as one who is accustomed to instruct others is expected to do. The illustrations are particularly clear and instructive. There is an absence of the usual galaxy of photographs of patients, "before and after," which goes with most articles that have been written on this subject. The author omits these photographs for the reason that they are often misleading and can be made to present results which are not in keeping with fact. This impresses the reviewer as the best treatise that has appeared on this recent field for the otolaryngologist.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Groningen, Holland.
Place: Madrid, Spain. Time: September 27-30, 1932.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. Walter G. Howarth, 21 Devonshire Place, W., London.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

Royal Society of Medicine, Section of Otology

President: Dr. Norman Patterson, 82 Portland Pl., W. 1, London.
Secretary: Dr. T. B. Jobson, Netherwood, Epsom Road, Guildford.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: Sept. 19-23, 1932.

American Bronchoscopic Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.

American Laryngological Association

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 2-4, 1933.

American Laryngological, Rhinological and Otological Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.

American Otological Society

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 2-3, 1933.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. Seventh St., Los Angeles.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. M. H. Gill, 36 Pearl St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Royal French, 28 S. First Ave., Marshalltown.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. W. J. Marshall, 212 N. Higgins Ave., Missoula.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Fort Worth. Time: Dec. 2-3, 1932.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

BUFFALO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: Third Monday of each month, September to May, inclusive.

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OTOLARYNGOLOGY

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

PITTSBURGH OTOLOGICAL SOCIETY

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS EAR, NOSE AND THROAT CLUB

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.